HOUSE OF LORDS

Select Committee on Science and Technology

Corrected oral evidence: The science of Covid-19

Tuesday 2 June 2020

10 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 Kakkar; Lord Mair; Baroness Manningham-Buller; Viscount Ridley; Baroness Rock; Baroness Sheehan; Baroness Walmsley; Lord Winston; Baroness Young of Old Scone.

Evidence Session No. 3 Heard in Public Questions 23 – 34

Witnesses

Dr Ellen Brooks Pollock, Lecturer in Infectious Disease Mathematical Modelling, University of Bristol; Professor Neil Ferguson OBE, Head of the Department of Infectious Disease Epidemiology, School of Public Health, Imperial College London; Professor Matt Keeling, Professor of Mathematics and Life Sciences, University of Warwick; Dr Adam Kucharski, Associate Professor, Department of Infectious Disease Epidemiology, London School of Hygiene and Tropical Medicine.

Examination of witnesses

Dr Ellen Brooks Pollock, Professor Neil Ferguson, Professor Matt Keeling and Dr Adam Kucharski.

The Chair: Good morning to our witnesses. Thank you all for helping us with our inquiry. It is much appreciated. In this inquiry, we want to test the science that drives the policies on the pandemic. We do not want to talk about what has happened, but to look forward at what the science can tell us about how we should manage the pandemic from now on, including a second wave if it comes, and subsequently. Your help in that respect will be most welcome. If something happens to my computer, Lord Mair will take over. We also have Jennifer Mills, who is working physically with the broadcasters. She will monitor those who want to ask supplementary questions. We have plenty of questions and the time for supplementaries may run out, but if I can get Members in, I will.

Q23 **Baroness Young of Old Scone:** Good morning, Dr Brooks Pollock and Professor Ferguson. First, what are the main types of models being used to model epidemics and pandemics, and how do they work? Secondly, how many models are fed into the Government's responses to the pandemic? How much have the results from the different models varied, and how have such variations been resolved?

Dr Ellen Brooks Pollock: Starting with the main types of disease models, the vast majority make the same underlying assumptions. Those are that individuals can be categorised by their infection state. In the most basic models, someone can be categorised as being susceptible to infection, or infectious and able to transmit the infection. You can make them more complex and add in extra infection states like asymptomatic infection or recovered and immune. That is central to the vast majority of models.

Models become more complex when we think about how individuals interact in populations: are all the individuals in a population in contact with each other at all times, or do we assume something a bit more sophisticated about who is in touch with whom, as well as how long people are in each of the infection states. For example, SARS-CoV-2 might have a latent stage when people incubate the infection for around five days. That is the basic transmission model.

Baroness Young of Old Scone: How many models have fed into the Government's response to this pandemic?

Dr Ellen Brooks Pollock: We have used a lot of different models. I have an example just from my point of view. I have been involved in six or seven different types of model looking at the different aspects of transmission ranging from a national-scale model of people mixing locally to an age-structured model, to a network model where connections are explicitly made between individuals. In total, there might be seven different groups and 40 different models, each looking at slightly different aspects of transmission.

Baroness Young of Old Scone: Presumably a number of different

schools of thought about models are still looking at the same set of issues but choosing to model them differently using underlying conceptions or data. Is that true, or is it not the case?

Dr Ellen Brooks Pollock: That is true, and I can give an example. I looked at the impact on the reproduction number for schools reopening. There were four groups using different underlying datasets to capture social contact patterns in slightly different ways. The models came up with slightly different results because of their underlying data, but overall the prioritisation of different interventions was consistent between the different models and the groups that contributed.

Professor Neil Ferguson: I think it is more relevant to talk about the number of different academic groups contributing to policy rather than the number of models. I run probably the largest group. We have had 50 people working on Covid using 10 to 15 different models, which are assigned for different purposes but are internally consistent. The SPI-M and SAGE process takes the different scientific viewpoints that are being expressed by contributing individuals and groups. Part of those differences and viewpoint approaches is down to model design and part to which data is selected and how it is interpreted. In these circumstances, it is quite difficult to separate a model from the group developing it.

Baroness Young of Old Scone: Dr Brooks Pollock said that some of the results are in the same ballpark, as it were, and demonstrate the same underlying advice that should be given to Government. Are there occasions when that is not the case and you get fairly divergent outcomes from the different modelling groups?

Professor Neil Ferguson: Yes. It is mostly down to the interpretation of the data rather than the model itself. Ellen referred to the issue of schools reopening. The fundamental uncertainty there is about how much children can transmit and how susceptible young children are to infection. The data is not clear, so depending on the assumptions you make you will draw different conclusions. Most commonly, where you see divergence between different modelling groups, whether it is significant or insignificant, it is down to how the parameters have been estimated and the different interpretations of the data.

Baroness Young of Old Scone: When these variations are significant rather than minor in the way Dr Brooks Pollock has outlined, how are they resolved by the scientific community in terms of its advice to Government?

Professor Neil Ferguson: It tends to lead to us saying that there is a lot of uncertainty depending on how the data is interpreted and thus drawing different conclusions, and really in trying to communicate the fundamental uncertainty. That can be addressed in some cases by collecting new data. For instance, the ongoing Office for National Statistics' prevalence survey looks at households and has addressed a significant data gap. The survey was set up about eight weeks ago partly because of exactly those types of discussion. We did not know how many people in the country were infected.

Q24 **Lord Winston:** Good morning, Dr Brooks Pollock, and it is very nice to see you, Professor Ferguson. I remember the evidence you gave to the Committee in 2006 on the reproduction rate of influenza when it was 1.8, and on the issue of travel and imports into the United Kingdom. What was intriguing was the surprisingly lower level of difference than would have been expected in that model. Which data do you choose to put into a model, and what are the parameters for that choice?

Professor Neil Ferguson: I will let my colleagues answer as well. It will vary by disease, but focusing on Covid-19 in the UK, early on we relied mostly on extrapolating data from China. We had very little data on what was going on in the UK. Basically, our indicators were on travellers; they were all we were looking at.

Surveillance in the UK has been ramped up and we now have a variety of data streams. None of them is optimal, and probably the most reliable is deaths from Covid-19. The difficulty with that is that people die up to three weeks after they are infected, so you are always looking back at what has been going on in transmission events. We have hospital-based surveillance, which is a little more timely, but again someone can be hospitalised 10 days after getting the infection.

We are now starting to get data streams that are more likely to give an understanding of what is going on in the community with the new expanded testing. Those are what we call epidemiological data. Adam might like to talk about other sources of data where we survey behaviour.

Dr Adam Kucharski: Following on from Professor Ferguson's comments, it is also really influenced by the question that we are trying to answer. If, for example, the question is, "What was the reproduction number in any given area a couple of weeks ago?", then, as mentioned, there would be several data streams—cases, deaths, hospitalisations, new infection data. Ideally, we would run analysis from all of them, and if we got the same estimate for R across them we could be very confident in our answer. If one dataset gave an outlier result, we would obviously have to try to understand what was driving that uncertainty.

If the question is more detailed—for example, what factors will drive the success of an isolation, test and trace scheme—we need much more information on what contact had been made in different settings and on the risk in different settings, and we would have to bring in a much wider range of data to try to inform those models.

Q25 **Lord Winston:** This question is for all of you. What differences are there between different viruses, for example other coronaviruses? What relevance does that have for our calculation of the reproduction rate and so on?

Professor Neil Ferguson: Immunity is the key thing with a new virus, but, with any pandemic, what defines the threat is that the human population has not seen it before. It may be more virulent, but there is also limited immunity. There is still a lot of uncertainty about how immunity works with coronaviruses, both the seasonal coronaviruses

where it appears that people have temporary protection against reinfection or at least disease, although that is not lifelong, and this coronavirus, about which we know even less. That is one of the key uncertainties at the moment.

Dr Adam Kucharski: Another aspect that is crucial for the ability to contain is how much transmission is occurring with clear symptoms. With SARS, a lot of the transmission happened when people had symptoms or were clearly ill. We are seeing with the new coronavirus that a large proportion of transmission happens before symptoms or potentially without, or with very mild, symptoms. That means that it is much harder for measures such as test and trace, which are triggered on people having symptoms, to ensure reduction in transmission. Essentially, as soon as you have a symptomatic case you are playing catch-up, because transmission may have already occurred.

Lord Winston: How do you decide which data to exclude from your model?

Professor Matt Keeling: I would be reticent about trying to exclude any dataset. All datasets will always be valuable. It is all about interpretation and understanding how that data can feed into the model, or rather how you can use the model to get an interpretation of what the data actually means. We are all looking at every piece of information that comes in, analysing it and trying to see how it fits to give us the best predictions.

Obviously, data from other countries has its own specificities. Equally, it has its own nuances, which are different from those of the UK. We have to be a bit careful with things like hospitalisations and that sort of thing from other countries, but in general we try to use as much information as is available to try to pass down the uncertainty that we have in any of the predictions.

The Chair: Dr Kucharski, you said that because the serial interval between a person getting infected and showing symptoms is rather long, it is much more difficult for strategies such as test and track to contain a disease. If I heard you rightly, that is what we are putting our hope in. What would be your comment on that?

Dr Adam Kucharski: The nature of the evidence that we have on transmission is that by the time someone has shown symptoms they have probably been infectious for a day or two already. That means that by the time someone has symptoms and reports it as a case, their contacts have potentially already been infected and those people may themselves become infectious, say three or four days further along—really very soon afterwards. That shows that, for these targeted test and trace measures to work, speed is of the essence, because as soon as someone becomes symptomatic you have a very short time window before their contacts may become infectious, and then you have another generation of transmissions to deal with. It really shows that speed is of the essence.

The Chair: Speed is of the essence to do what?

Dr Adam Kucharski: To isolate the initial case, trace their contacts and quarantine those contacts.

Q26 **Baroness Walmsley:** My questions are about assumptions. Perhaps Professor Keeling might start off and then we might hear from Dr Kucharski and others. What assumptions do epidemiological models make? Right at the beginning, in the early stages of this pandemic, when we knew very little about the virus and its transmission, how did you determine what these assumptions should be? Can you tell us how these assumptions have been refined over the course of the pandemic, particularly how they might have changed in the UK over the course of February and March this year?

Professor Matt Keeling: As Ellen said at the beginning, most models are based on a standard assumption that we move through various compartments, starting off as susceptible, through a latent or exposed class, then becoming infectious and finally recovering. In general, that framework, together with age structure, tends to be very good at predicting almost all outbreaks. We are now at the stage where we have a good understanding and from relatively sparse data can at least start doing early approximations of quantities such as how quickly the outbreak is growing, because we are fitting to these type of data.

One of the biggest problems is that there is a lot of uncertainty in the details. Whilst we are able to predict the early phase of the outbreak and its growth very well, as you start reaching the peak, whether it is due to control measures (or it being a different outbreak where you may be using up all the susceptibles in the population) predictions become much harder and there is much more uncertainty.

We have obviously brought in a lot of understanding from SARS, which is another coronavirus. There are some similarities there, which helped at least in the early stages. We also knew from some of the Chinese data that there was probably a significant number of asymptomatic individuals who were potentially passing infections. That was something else that was added to the standard structure.

As for what has changed, early on there was huge uncertainty, and there still is quite a lot, about the role of asymptomatic individuals. Thinking about what has changed, we probably over the first few months got a much better understanding of hospitalisations within the UK—how to translate infectious cases within the model into those that go to hospital and how long they stay there, and the fatality rates. Just bringing that source of data from the UK helped to refine things and make the models much more accurate, although obviously in the early stages we were all using data from Wuhan and the cruise ships that were coming in, because that gave us probably the earliest signals.

Baroness Walmsley: Because we were so far behind China, Italy and the cruise ships, to what extent did that information change the assumptions you made?

Professor Matt Keeling: Most of the models started once data was already available from Wuhan, so that did not change much. The biggest

changes were actually in trying to understand the situation in Italy. It was unclear in the early stages of the Wuhan outbreak whether we would get a similar scale of outbreak in the UK or elsewhere in the world. Italy was the big eye-opener, where we realised that we could have a large outbreak in the UK. I think that started changing our thinking, but throughout it has been a gradual refinement rather than a eureka process of suddenly changing everything and the models changing dramatically.

Baroness Walmsley: As you went along, did you perform sensitivity analyses on any of these assumptions?

Professor Matt Keeling: Yes. Sensitivity analysis usually involves thinking about changes to parameters. I think most of us are using what is known as a Bayesian framework, so the uncertainty in parameters is already built into the model. Our model takes that uncertainty in parameters and percolates it forward in time, so we are always looking at uncertainty as we move forward.

But we also think about model uncertainty. If we change some of the fundamental questions and assumptions in the model to how the disease is actually progressing, does that change our results? Very often, given that we are already constrained to match the available data, the changes are quite mild.

Baroness Walmsley: Thank you. Do any of the other witnesses want to come in on that?

Dr Adam Kucharski: As mentioned, we were trying to draw on all the available data sources that were there. In late February, for example, we did a meta-analysis of all the severity estimates that were coming out, particularly from China, and estimated that about 20% of hospitalised cases would have acute respiratory distress syndrome and about 15% would require ventilation. That is a measure of likely ICU requirements. Obviously, there was quite a lot of uncertainty around that, and as we got more data in from other countries we could start to narrow that.

Similarly, with other key metrics like the infection fatality rate, even quite early by pooling datasets, we did some analysis and Imperial independently put together some estimates suggesting that probably between 0.5% and 1% of infections would be fatal. Obviously, as more data comes in, we can then see how those estimates are holding up, and what might have started with quite a large uncertainty range will narrow.

The Chair: Going forward, Professor Ferguson, what do the models tell us?

Professor Neil Ferguson: That is a very large question. Coming back to the question just asked, one of the things that we learned from Spain and Italy in early March, or soon after that, was that the UK had been much more heavily affected than we had anticipated. That is one reason why we have if not the largest then one of the largest epidemics in Europe.

Going forward, the models say that we have limited room for manoeuvre and that this is a highly transmissible pathogen. We have reduced transmission by about 80%, but to maintain control we need to keep that

transmission suppressed by about 65% or so, so we have a little bit of wriggle room. It will be a learning experience as to how we allow society to resume whilst maintaining control of transmission.

Baroness Walmsley: May I ask about the granularity? We have learned a lot of detail as the pandemic has gone on about the sorts of people who are most badly affected. I am thinking of people with obesity, the BAME population, and so on. Have those bits of knowledge made any changes to the assumptions that you are making as we go along?

Professor Neil Ferguson: As Matt and Adam said, we feed in that data particularly when modelling the health impact of the epidemic, but it does not necessarily make a change to the fundamental assumptions about transmission.

Yes, the BAME population has been very badly affected, but it is not clear whether ethnicity is an independent risk factor or is correlated, frankly, with socioeconomic status and the ability to shield; we have clearly seen in this epidemic that the middle classes and the wealthy are much more easily able to isolate themselves than people who have to go out and do essential jobs. There are also risk factors like cardiovascular disease, and obesity as you mentioned. Some of these risk factors have higher prevalence in BAME groups. It is a very complex picture.

Q27 **Baroness Sheehan:** How do the models account for uncertainty in individual behaviour, despite population-level regulations?

Dr Ellen Brooks Pollock: The models range in complexity from simple models that do not capture much individual variation to more complex models. We have been using social contact data that tries to capture individual variation to look at the kind of impact that has. It varies by model complexity. Essentially it is the data that feeds into the model that enables you to capture these individual variations.

Baroness Sheehan: Could the other panellists answer that question, as well as this one? The public's adherence to lockdown appears to be better than expected, and as evidence of this emerged did it inform the models' assumptions?

Professor Neil Ferguson: Maybe I can comment on that. In late February/early March, we were all modelling the potential effect of social distancing and other measures. We basically had to make assumptions about what population adherence would be, because we did not really have any data. You are right that we saw greater adherence than was expected, which immediately fed back into the models.

Professor Matt Keeling: I would echo that. A lot of the models are being fitted to epidemiological data—hospitalisations and bed occupancy—so it is very difficult to directly translate adherence to the lockdown measures straight into something that tells you about transmission. If we know that 85% of people are obeying the lockdown, that does not immediately tell us what the drop in R is, so we also have to factor in data from hospitals, deaths and other factors that tell us about how that drop relates to an epidemiological change in transmission.

There is a two-step process there. So, although that information is incredibly useful and very quickly allows us to say that our early predictions may have been too pessimistic, we still lack the information to know how to translate adherence directly into transmission.

Baroness Sheehan: Would you say that the public's adherence and the assumption early on that it would be quite low affected policy by decision-makers quite substantially, given that they delayed lockdown because they felt that fatigue in isolation would set in? I just wonder why that assumption about people's behaviour was so different from what turned out to be the case.

Professor Neil Ferguson: First, the issue of fatigue, which we discussed, was not something that we ever modelled. Some people in SAGE had that view, but it was not one that I shared or other modellers looked at. I think the difference in adherence was because we assumed that, for instance, there would be a 75% drop in contact outside the home, and there is a benefit to being conservative when you are looking at how effective the policy will be. It turned out to be more like an 85% drop. So we are talking about differences but not ones that make a qualitive change to what you predict a policy to do.

Dr Adam Kucharski: We considered a range of reductions in a model, such as changing the parameter so that you have a 50% reduction in outside contact; in one scenario, we looked at a 90% reduction. Likewise, with some of the more recent contact tracing analysis of the adoption of an app, optimistically we saw that perhaps half the population might end up having it. That can present the scenario that, if you manage to achieve that, it gives an indication of what transmission might be, but obviously there is a much wider set of questions about how you might do that and what the messaging and policy might be that ends up giving you that value.

Q28 **Baroness Sheehan:** Thank you. I want to finish by asking a question about the R value. How is uncertainty in R0 taken into account in models? How sensitive are models of different interventions to different values of R0?

Dr Adam Kucharski: We used a range of values of R0 in a lot of our scenario analysis. In early March we used values ranging from 1.5 to 4, which, from early data, was the plausible range we would see. Some suggestions about numbers of cases would be dependent on the reproduction number, but in many cases the relative impact of interventions may be less so. For example, we recently looked at isolation, contact tracing and social distancing. The relative ordering of the effectiveness of those is less sensitive to R, but the exact reduction is more dependent on what you assume.

Viscount Ridley: I have a supplementary question on this. A number of mathematical analyses have concluded that the lockdowns may not have had such a big effect as we are thinking, because the trajectory was already beginning to bend because of voluntary measures—no large gatherings and things like that. In a recent study of the lockdowns in the

UK, Spain, France and Italy, Thomas Meunier, a mathematician in Canada, said, "Comparing the trajectory of the epidemic before and after the lockdowns, we find no evidence of any discontinuity in the growth rate, doubling time and reproductive number trends". He goes on to conclude that lockdown may not have saved any lives. Would any of the panel like to comment on that?

Professor Matt Keeling: I would strongly disagree with that. All our modelling suggests that the lockdown had a major impact on the growth of the epidemic. There is a very clear step change. One of the difficulties with trying to analyse this is the huge range of delays in the system. You may do a lockdown and have a change in policy one day, but you are not going to see the impact of that in hospitalisations for at least a week, if not longer. Very often, these things may take one or two generations to cycle through the population. If you suddenly do a lockdown and people have stronger interactions within their household, that may keep the epidemic going for another generation, so it could be three or four weeks before you see the impact of any lockdown. You have to be very careful with some of these analyses.

Viscount Ridley: Would that not work in the other direction? In other words, if the lockdown does not have an effect for several weeks, you would think that the peak would come even later, yet it came quite early in April.

Dr Adam Kucharski: There is a slightly counterintuitive thing going on here. If you have a sudden drop in transmission, most of your burden of infection occurs before it. Because you have this distributed delay, you would not have a drop in infections and see a simple drop in deaths three weeks later; it would actually spread it and you would get a peak earlier. As mentioned, a lot of the analyses that have rigorously accounted for these delays can reproduce the shapes that we have seen. It is not just a simple case of moving the curve along by a few weeks.

Q29 **Baroness Rock:** I would like to explore the different models a bit more, if I may. Were there any significant differences between the models being used by SAGE? If so, what were they? Were there more optimistic scenarios and some reasonable worst-case scenarios from the different groups?

Professor Neil Ferguson: The reasonable worst-case scenario is a government scenario. It is determined as a consensus judgment of exactly that—what is a reasonable worst case for government to be planning to. The exact model that has been used to generate the reasonable worst case has varied over time. Right now, it is Matt Keeling's model that has generated the current reasonable worst-case planning scenario. Those scenarios are updated through time. They are based on data, but they are deliberately intended to be challenging for the Government to plan to.

The challenge that we had in early March, though, was ending up with a reasonable worst-case scenario that in my view was very close to the best estimate of what would happen. That can lead to a slight disconnect

in thinking. In terms of policy making, I think the view was almost, "This is the reasonable worst case. We're not going to see anything quite as bad as this". John Edmunds and I were not quite so sanguine about that.

There are always differences between modelling groups, so the precise estimate of the reproduction number and the infection fatality ratio will vary, but they will always be, and have been throughout this pandemic, similar enough for there to be a fair degree of confidence that these would not be qualitative differences for policy conclusions.

Professor Matt Keeling: I would echo that. Yes, there were differences, but in general we work very closely to try to resolve them. SPI-M, one of the subgroups of SAGE, has been incredibly helpful with that; we look at the outputs of all the different models weekly—more frequently early on—and try to reach a unified scientific consensus. This is not just about taking an average but about trying to understand the differences as well. It is very much a judgmental opinion, based on taking all the evidence from the different models and looking at all the predictions.

That has been incredibly helpful throughout. As Professor Ferguson said, most of the differences are relatively minor and they come mainly from the different assumptions and data feeds that are used. It is questions and nuances like how much you decide to trust hospitalisations versus the mortality rate that really influence things.

Baroness Rock: Thank you. Can the panellists comment on the differences between modelling approaches undertaken in the UK and in other countries?

Professor Neil Ferguson: I can comment, because we are supporting and working with a lot of other countries. I do not think there are huge differences. There are big differences by country in how science interfaces with policy. Some other countries are using models we have generated, but the ones generated in France, Germany and the United States are qualitatively very similar and are coming to similar conclusions.

The Chair: Are you saying, Professor Ferguson, that the use of science is the same in every country, but the policies are different? The example you did not quote was Sweden.

Professor Neil Ferguson: I have the greatest respect for Anders Tegnell and the scientists there. They came to a different conclusion but based on quite similar science. They make the argument that countries will find it very hard to stop second waves. Therefore, by front-loading the health impacts, their policy will allow them to leave lockdown earlier. I do not agree with that, but scientifically they are not that far from scientists in any country in the world.

Professor Matt Keeling: I agree. We would hope that the science and the predictions from all countries would be very similar. The fundamental underpinning of the SEIR models, that we have all been talking about, is the compartments that we put people in. So, despite the differences in the data and population structure we would expect very similar science and predictions to be coming out of all the countries. The interpretation is

a value judgment about what you believe will happen in the future. If you believe that a vaccine is coming soon, you want to clamp down on the first wave as much as you can. If you believe that we have to get through this one way or another, you may decide to front load, as Sweden has done. It is really about how you do the trade-offs between economics and epidemiology, and where you put the inference.

Dr Ellen Brooks Pollock: Although we are talking about the similarities between the different models, it is hugely valuable that there are multiple different models. Often the differences between the models have been quite insightful about what the important processes are. It is essential that multiple models contribute to what is discussed at SPI-M rather than evidence for policy being based on a single model.

Viscount Ridley: I am surprised by what Professor Ferguson said about Sweden. I would like to come back to that before I move on to R and R0. Uppsala University took the Imperial College model—or one of them—adapted it to Sweden, and forecast deaths in Sweden of over 90,000 by the end of May if there was no lockdown, and 40,000 if a full lockdown was enforced. In fact, there have been only 4,350 deaths in Sweden to the end of May.

This is a huge discrepancy and suggests that something was wrong with the model. We have heard today that the models are thought to have performed well on the whole. It does not sound to me like they have performed well. But we are looking forward.

Professor Neil Ferguson: May I quickly respond to that? First, Uppsala did not use our model; they developed a model of their own. We had no role in parameterising it. Generally, the key aspect of modelling is how well you parameterise it against the available data. To be absolutely clear, they did not use our model; they did not adapt our model.

Viscount Ridley: But surely the key point is that, from what we have just heard from both Professor Ferguson and Professor Keeling, without a lockdown one would expect a relatively high death figure in Sweden. In fact, it is much lower that the UK.

Professor Neil Ferguson: That is an interesting question. It is clear that there has been significant social distancing in Sweden. Our best estimate is that that has led to a reduction in the reproduction number to around one.

It is clear when you look at Sweden's mortality that it is not seeing the rate of decline that most European countries are seeing. Nevertheless, it is interesting that adopting a policy that is short of a full lockdown—secondary schools and universities have been closed and there is a significant amount of social distancing, but it is not a lockdown—has gone quite a long way towards the same effect, albeit that there is no evidence of a rapid decline in mortality as there has been in other European countries.

We are looking at this very closely. Lockdowns are very crude policies. Going forward, we would like to have much more targeted control of transmission, which does not have the same economic impacts.

The Chair: Viscount Ridley, would you like to move on to your main question?

Q30 **Viscount Ridley:** My question is about R and R0 in particular; it zeros in on one aspect which I find puzzling.

The SAGE minutes from 20 March show that SAGE knew then that a major problem was nosocomial infections—that is, hospital acquired. Given a very high and rapidly changing R inside hospitals and care homes, and a much lower R in the community, was there ever any prospect of an overall average estimate of R being useful?

Dr Adam Kucharski: It depends on what you want that R estimate to give you. The standard usefulness of R is that is tells you what your case burden and hence your fatality burden will be in the coming weeks. If your cases are flat, you will see the implications of that in the coming weeks, regardless of where cases are happening. In that sense, an overall R is useful.

If you are talking about targeting control measures, you need that finer-scale data on exactly what settings are driving transmissions. Obviously, more recently, we have seen a lot more transmission concentrated in care homes and healthcare. This needs to feed into discussions about how we are targeting control measures. It comes back to what you are using those R values to try to estimate.

Viscount Ridley: But we heard last week from one of our witnesses that transmission in the community may have been very low because of the lockdowns and other measures, and that most of the cases that are happening now, even in the community, are coming back out of the hospital and care-home settings. So, in a sense, a high R in one part of society is keeping R slightly higher than it would otherwise be elsewhere in society. This heterogeneity of ours seems, to me certainly, to be underestimated in policy conversations and possibly in modelling.

Professor Neil Ferguson: I agree. That hypothesis is the one that we have been considering a lot in recent weeks. It is the most likely one to explain why R is hovering just below one.

There has been modelling of hospital transmission. It is quite hard to build a model that includes both hospitals and the community, and is parameterised reliably. But that nuance is understood in the interface between science and policy. Like many people, I am shocked by how badly European and other countries around the world have protected care home populations. However, that is a slightly different issue from science.

The Chair: Going forward, what does that tell us?

Professor Neil Ferguson: If we did a better job of reducing transmission in closed institutions like hospitals and care homes, we would have a bit more wiggle room.

These epidemics are not completely separate from one another; they are coupled. As Lord Ridley said, the infections in care homes and hospitals spill back into the community, more commonly from the people who work

in those institutions. If you can drive the infection rates low in those institutional settings, you drive the infection lower in the community as a whole.

Q31 **Baroness Manningham-Buller:** All witnesses have told us that some of this is pretty difficult because we have incomplete information and uncertainty about the virus, although we are learning the whole time. Looking back at the beginning, there were some quite worrying assumptions about the death rate. I am not saying that the modelling was alarmist, but some worrying data was coming out.

Looking back, how much do you think we got right, how much did we not, and why? I very much recollect Patrick Vallance saying that 20,000 deaths would be a good result. It seemed at the time to be a deeply shocking remark, but we are now at nearly double that.

What is the explanation of the gap between that early modelling and where we are today?

Professor Matt Keeling: We are modelling a constantly evolving situation, so we can make predictions only from the data that is currently available. In the early stages, in the absence of a lockdown, the very alarmist values coming out were about a worst-case scenario. If we just let the epidemic run, how bad could it be?

That is the sort of information that policymakers needed at that time. Your comment about the 20,000 deaths if things went well has been borne out. The lockdown could have been very strict. We could have thought more about what was happening in care homes and hospitals early on, as you have heard. That might be one area where modellers did drop the ball.

With hindsight, it is easy to say that we know that care homes and hospitals have these huge collections of very vulnerable individuals, so maybe we could have modelled those early on and thought about the impacts there. But considering the amount of information that we had at the time, the models offered the best estimates of what could happen in the short term. Long-term predictions are much, much more difficult. Very often, our models have quite large confidence intervals, but only the mean is reported, so a whole raft of different elements need to be brought into this.

Baroness Manningham-Buller: A supplementary question might be what has surprised you between the predictions that were based on possibly incomplete information in February and March and where we have got to.

Professor Neil Ferguson: I mentioned earlier that one thing the genetic data is showing us now is that most chains of transmission still existing in the UK originated in Spain and to some extent Italy. We had been worrying about the importation of infection from China. We are a very well-connected country in the world, particularly with Asian countries and maybe the US, but it is clear that before we were even in a position to

measure it—before surveillance systems were set up—many hundreds if not thousands of infected individuals came into the country in late February and early March from that area. That meant that the epidemic was further ahead than we anticipated. That explains some of the acceleration of policy then, but it also explains to some extent why mortality figures ended up being higher than we had hoped.

Dr Adam Kucharski: Early on as well, we had pretty good evidence that what China had implemented had suppressed its transmission and that, if you were to introduce only light measures and sit back, your health systems would quickly be overwhelmed. We had empirical evidence from Italy that that was the case. The uncertainty was very much in the middle of all the measures that were coming in as part of lockdown. Obviously, lockdown in the UK has not been the same as in Italy or China; it is really a basket of interventions. As countries lift measures, we are seeing a variation in the order and the timing at which they do that, which reflects how much we still need to learn about how much each of those has contributed to control.

The Chair: Looking ahead, accepting that there are wide confidence limits in the models, what in the short and medium term are the models saying is likely to happen now?

Professor Neil Ferguson: One of the key issues is how much relaxation of current controls will lead to potential increases in transmission. I should say that it is not clear that it will automatically do so. Most of the modelling would suggest that we might get a small increase, but it is very unclear whether that would lead to the reproduction number going above one and there being a bump in the rate of infection or whether it will stay just below one, so there is a lot of uncertainty right now.

I suspect that in any scenario, levels of transmission and numbers of cases will remain relatively flat between now and September, short of very big policy changes or behaviour changes in the community. The real uncertainty is what will happen if there are larger policy changes in September and we move into the time of year when respiratory viruses tend to transmit slightly better. That remains unclear.

Professor Matt Keeling: The only thing I would add is that we are assuming that adherence and changes to the lockdown rules apply to the UK or England as a whole, depending on whether it is a national figure or not. If there are sections of the community that start adhering less well, we could see more pockets of infection arising. That is another area of concern, and one that would be hard to model because we would not have those individual-level behavioural details that we mentioned before.

Q32 **Baroness Blackwood of North Oxford:** I am interested in the intersection between modelling and measurement. As we ease the lockdown and introduce test and trace, I wonder whether that will enable us to estimate more effectively variations in transmission—the K rate—and whether we will have more accurate transmission, which will enable a more sophisticated policy measures at a local level.

Dr Adam Kucharski: That would certainly be useful. The more data we have the better. A lot of our understanding of which settings and which interactions drive transmission come from detailed contact tracing studies in other countries. South Korea and its extensive testing around clusters gives a lot of insight into how transmission is happening, and in turn that can inform refined control measures. The hope is that if we have detailed surveillance and increased testing it will give us a more accurate picture of where and how transmission is happening. In turn, that can inform potentially more effective and less disruptive control measures.

The Chair: Increased testing of whom?

Dr Adam Kucharski: As a baseline, increased testing of everyone with symptoms, but also studies that follow up individuals through contact tracing who are potentially exposed. Testing of people at risk can give insights into the sorts of settings and interactions that might be driving transmission.

Baroness Blackwood of North Oxford: Would that include screening of those in areas of high rates of infection such as care homes and NHS settings?

Dr Adam Kucharski: That would be useful. Some of the data that has been coming out of UK hospitals, where there has been widespread screening, has given us some idea of what the prevalence of asymptomatic infection might be. So testing in those high-risk environments, as well as having implications for control, can help us to get at cases that have very little or no symptoms and how they are potentially contributing to transmission.

Q33 **Baroness Young of Old Scone:** How much modelling has there been, of varying degrees of success, of the test and trace regime as it goes forward, and how widely variable are the outcomes if there is greater or lesser success?

Professor Neil Ferguson: We, Matt Keeling and Adam have all looked at different likely policy effectiveness. It is not a panacea, for the reasons Adam and others have given. It depends not just on what proportion of people show symptoms, but on what proportion of people can actually identify contacts and how quickly those contacts are identified and in what proportion they then enter into isolation. We are asking people a lot to isolate for 14 days. The modelling has reflected that. We have done a lot of sensitivity analysis. If it works perfectly, we think that it might reduce the reproduction number by 0.2 or 0.25, which is significant but not huge. But it could be less than that.

Q34 **Lord Borwick:** Professor Ferguson, I do not trust the newspapers, but one or two of them have suggested that the original Imperial model was written in Fortran, a rather elderly program. Was there any truth in that?

Professor Neil Ferguson: No. It was written in C, which is also an old programming language, but one that is very actively used today. We have just released the files, which allow people to reproduce the results in our *Report 9* so people can check themselves.

Lord Winston: My question has been answered.

The Chair: Does anybody else have any supplementaries? If not, I will close this session.

Thank you all very much indeed for helping us. It has been a most interesting session.