HOUSE OF COMMONS

Science and Technology Committee

Oral evidence: UK Science, Research and Technology Capability and Influence in Global Disease Outbreaks, HC 136

Wednesday 10 June 2020

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Watch the meeting

Members present: Greg Clark (Chair); Aaron Bell; Dawn Butler; Katherine Fletcher; Andrew Griffith; Mark Logan; Carol Monaghan; Graham Stringer; Zarah Sultana.

Questions 784 - 905

Witnesses

I: Professor Mark Woolhouse, Professor of Infectious Disease Epidemiology, University of Edinburgh; and Professor Johan Giesecke, Former State Epidemiologist for Sweden and Professor Emeritus, Karolinska Institute.

II: Professor Neil Ferguson, Professor of Mathematical Biology, Imperial College London; Professor Matt Keeling, Professor of Mathematics and Life Sciences, University of Warwick; and Dr Nicholas Davies, Research Fellow in Mathematical Modelling, London School of Hygiene and Tropical Medicine.

Written evidence from witnesses:

– [Add names of witnesses and hyperlink to submissions]

Examination of witnesses

Witnesses: Professor Woolhouse and Professor Giesecke.

Q784 **Chair:** In this session we will consider questions about the use of scientific modelling during this pandemic. We are very pleased to welcome our first two witnesses. Professor Mark Woolhouse is Professor of Infectious Disease Epidemiology at the University of Edinburgh. He sits on the Scientific Pandemic Influenza Group on Modelling, known by the acronym SPI-M, and the Scottish Government's Covid-19 Advisory Group.

Professor Johan Giesecke is Professor Emeritus of the Karolinska Institute in Sweden and former chief scientist at the European Centre for Disease Prevention and Control.

Thank you both for appearing.

Perhaps I may start with a couple of questions to Professor Woolhouse. Professor Woolhouse, you are a member of SPI-M. You see the policy actions and now the minutes of SAGE to which the group reports. How influential would you say epidemiological modelling has been in response to the pandemic?

Professor Woolhouse: I would say the same thing as the chief scientific adviser says. It has been very useful and influential, but it is not the only strand of evidence that goes into Government decision making, nor should it be.

Q785 **Chair:** On 23 April you wrote, "I do think scientific advice is driven far too much by epidemiology—and I'm an epidemiologist." Can you explain what you meant by that?

Professor Woolhouse: Yes, I can. I was particularly concerned that we were looking at only one side of the equation when assessing the costs and benefits of lockdown. There has been a lot of emphasis on the public health burden of Covid-19. In the early stages of the epidemic, before we had large amounts of data, that was largely on the basis of modelling, and that is all right and proper and as it should be, but we are looking literally at only one side of the equation when we do that.

The other side is the harms done by lockdown. By those, I mean the harms in reduced access to healthcare provision, which has been very marked during this epidemic, the harms to our mental health and social wellbeing, the education of our children and our economy. It seems to me that a balanced assessment of the merits of lockdown requires both sides of the equation to be modelled. We were looking only at the public health side.

Q786 **Chair:** How do you know that predominantly one side of the equation was being looked at in the advice SAGE was then giving to Government and Government then acted upon?

Professor Woolhouse: I am sure it was not the only one that had been looked at, but it was the only one I know of that was quantified by modelling where the results were published in full quantitative detail. I have not been able to assess any of those other harms because I have not seen a thorough and comprehensive analysis of them. If it exists, I am very happy that it does, but I have not seen it.

Q787 **Chair:** On the use of models generally, the Committee took evidence last Friday from Professor John Kay, who thought that the most important role of modelling was not so much to make point predictions—or even a range of predictions—of outcomes, but to understand the key variables and what is driving a phenomenon. What would you say to that view?

Professor Woolhouse: I very much agree with that view. I do not think it was ever possible to predict the course of this epidemic; there were too many fundamental unknowns to do that, and the work of my group does not attempt to do that. What we do is explore a wide range of possible scenarios—all, we hope, plausible—and try to understand, exactly as suggested by your witness last Friday, how the epidemic is likely to behave under a series of different assumptions and interventions and explore the whole range of those. We never make a single prediction.

Q788 **Chair:** Is it your view that we have been too reliant on predictions for the response, in so far as you have been part of discussions that have fed into SAGE, which have fed into ministerial decisions?

Professor Woolhouse: I cannot judge how much individual predictions vary, but there is a lesson for us in communicating the outputs of a modelling exercise. It is very important that we give a range of possible outcomes, modelled in the context of different scenarios that are carefully spelled out, before making a judgment about what is likely to happen in the future without having a single prediction. I do not think that is helpful.

Q789 **Chair:** As for the other side of the equation, we do not know what the advice has been to Government. We have recently seen minutes of SAGE meetings, but those are minutes rather than the actual advice to Government. Does looking at the minutes give you any insight into whether the balance in the equation, as you put it, has been right in SAGE discussions?

Professor Woolhouse: I am not on SAGE, as you know. SAGE has a broader range of inputs, but I think I can speak more from my experience on the Scottish Advisory Group, which is sometimes informally known as SAGE for Scotland, where there is some discussion of both sides of the equation, but again not the full quantitative analysis. The full quantitative analysis always comes down on the epidemiological and public health side, so I do think there is an asymmetry there.

Q790 **Chair:** Professor Giesecke, you have observed what has been going on in the UK and other countries. How typical is the approach that has been taken in the UK to modelling during the pandemic in other countries?

Professor Giesecke: It is very strong in the UK and has been for a long time. Excluding the US, it is probably the country with the strongest modelling tradition. Could I answer the first question you asked Professor Woolhouse?

Chair: I would be delighted.

Professor Giesecke: I could not agree more with that. I think epidemiologists and modellers had too great a power in your country as well as Sweden in the earlier phase of the epidemic. I say that as an epidemiologist. Decisions that were political were to some extent left to the public health agency, and in a way the Government just signed the paper. I am exaggerating a little, but to some extent we have the same problem. You had another question, not the one I answered.

Q791 **Chair:** I am grateful for that answer. To bring the two questions together, from your observation of other countries, have you seen what Professor Woolhouse described as a more balanced approach in other countries' disciplines? How has it been in other places?

Professor Giesecke: I think there has been a more balanced approach. I think many countries—I am talking mainly about European countries because those are the ones I know—looked to the UK and followed it to some extent.

Q792 **Chair:** Tell us how the advice to Government has been formed in Sweden.

Professor Giesecke: The public health agency, which has epidemiological expertise, is very close to the Government. It meets Government representatives twice a week or so. To take one example, we never closed schools in Sweden. From age zero to 16, children were still at day care or in school. That decision has enormous economic ramifications, because about 10% of the population of Sweden is below the age of 10 and needs someone to look after it. That meant at least one parent had to stay home with their child if you closed the school or day care. The cost of that was to some extent looked at in the Public Health Institute, but to me it is purely a political decision.

Q793 **Chair:** Did the scientists advising the Public Health Institute agree with the policy decision that was taken, or is it known what their opinion was?

Professor Giesecke: They suggested it—the decision to keep schools open.

Q794 **Graham Stringer:** Professor Woolhouse, I ask a slightly philosophical question. You agree with Professor Kay's analysis that models are useful for seeing how variables will react with each other. The Government say regularly that they are following the science, but does the advice that is coming from modellers pass the Karl Popper test of being scientific? He said that what separates science from all other activities is the ability to falsify it. I cannot see how these models can be falsified.

Professor Woolhouse: Thank you very much for the question. It is very philosophical, and the answer is yes. The models can be falsified in many ways and that is what we do as part of the validation exercise for models. The first and most obvious way is that the models make predictions and the predictions either come true or are false. Back in 2001, in the foot-and-mouth disease epidemic, my group predicted that the epidemic would double in size in the next eight days, counter to the advice the Government were then receiving. That prediction came true. The model passed that test, but it may not pass further tests. That is how science progresses.

Q795 **Graham Stringer:** Do you think the models the Government used this time have passed critical test, because in many ways we will not know whether they have passed those tests for some time?

Professor Woolhouse: That is a very good point. I think it is important that we continually attempt to validate our models in the best ways we can. The example I gave to the Lords Select Committee last week was that the very simple models we have been using do not use the data on serology, which give you a marker for the number of people who have been infected. Therefore, we test our models against whether they are capturing the patterns in that. My understanding from the models in SPI-M is that most of them capture those data quite well, even though in the initial stage of the epidemic they were not part of the input.

That is an internal validation, but you are quite right. There will be a lot more validation to be done in retrospect than can be done.

Q796 **Graham Stringer:** Quite a considerable burden has been placed on the models and modellers, when really the Government were faced with two problems: first, they worried that intensive care units were going to be overwhelmed by patients; and, secondly, they did not believe that Public Health England could provide the tests necessary for a track and trace system. From what you have observed, do you think that the decision to go to lockdown was based mainly on the worry about intensive care units?

Professor Woolhouse: That was certainly a factor playing in. I always understood when I was sitting on SPI-M at the time that Government policy was threefold: saving lives, protecting NHS staff and avoiding the NHS being overwhelmed, as you just described.

Q797 **Graham Stringer:** On the last point you made about saving lives, the statistics will not be available for some time, but, on the point you made at the very beginning in answer to the Chair's questions, the way we have focused on hospitals, making sure intensive care units work, could cost more lives than it has saved. Do you think that is a reasonable hypothesis?

Professor Woolhouse: I do not think I fully understand the basis of that statement. You will have to enlighten me.

Q798 **Graham Stringer:** It is a question, really. The objective of the lockdown, putting a lot of effort into making intensive care units work, was to save lives, as you said, but when you take into account the economic impact and the impact of effectively switching off other parts of the national health service so they are not easily accessed do you think it is a reasonable hypothesis that, net, lives will not have been saved?

Professor Woolhouse: I am going to broaden that out slightly. I do not think we will able to do a full reckoning of the cost of lockdown for many years yet, but I have no doubt that lockdown will cause loss of livelihood, wellbeing and quite possibly a lot of lives, but we will not be able to balance that out for some time.

We saw that very clearly during the Ebola outbreak in west Africa a few years ago. The indirect effects of Ebola on the health systems in that region were considerably worse than Ebola was, and that was bad enough. I fear that to a degree in the UK, but certainly internationally—for example, Africa—the costs of lockdown may be considerably worse than the disease itself.

Q799 **Aaron Bell:** Perhaps I may begin with questions to Professor Giesecke about uncertainty in these models. There are inherent uncertainties in all models, but what do you think are the most important uncertainties in the models we have? What things do you think are not being picked up by the models we have used so far?

Professor Giesecke: There are two things. One is the number of cases that are not diagnosed. I would not use the word "asymptomatic" because many of them can be quite sick. I had friends who were really sick, but they never came into the healthcare system and were never recorded. That proportion of sick people has been underestimated by many models.

The second is the case fatality rate, which was severely overestimated at the beginning of the outbreak in most models, and that happens in any outbreak, as Professor Woolhouse well knows. We almost always overestimate the severity of a new disease. There are many examples of that, but with time we learn that more and more cases, which were never detected originally, were cases. I think those are the two things.

Q800 **Aaron Bell:** Given the case fatality rate, what do you think the implications were for the decisions that the UK and other countries took at the beginning? Obviously, Sweden took a different course on that. Was that informed by your suspicions about the case fatality rate, or was it based on other assessments?

Professor Giesecke: To some part, yes. We felt that the denominator used to calculate the CFR was too small. There were more cases that were not detected, which would bring down the death rate.

Q801 **Aaron Bell:** Modellers attempt to deal with uncertainty by conducting sensitivity analyses. How useful do you think those are in removing

uncertainty from models, or does it give us a false sense of precision?

Professor Giesecke: To some extent it gives a false sense of precision, because in most of the models I have seen there are so many assumptions. You can change one back and forth and change another back and forth, but it is a complex web—a complex net—and it does not really respond to these sensitivity analyses.

Q802 **Aaron Bell:** Professor Woolhouse, do you have any comments about the uncertainties and the things the models may have been missing then and now?

Professor Woolhouse: I agree with the comments Johan just made. With respect to the case fatality rate, one thing that was not immediately apparent from the data emerging from Wuhan in China in January and February was the very marked disparity of case fatality with age. It is extremely marked. This appeared in a number of publications during February, so by the time the disease hit the UK this was known.

The simplest way to put it is that the burden of this disease is vastly concentrated in the elderly. Eighty per cent. of deaths in the UK so far have been among the 20% of the oldest population. So far, if you are over 75 in the UK, the chances of dying, compared with a child under 15, have been 10,000 times higher. These are very marked disparities. In my mind, that ought to influence the public health response and put the public health response where it is most needed. This is very predominantly a disease of the elderly.

Q803 **Aaron Bell:** Looking at the functions of the SIR model, on which most of the models are based, it seems to me we have issues at both ends of the model. We do not know enough about what immunity is conferred, or for how long, which affects the people in the R capacity, and there also seems to be some dispute about how susceptible the population is. Most models assume the whole population is susceptible, but we seem to be getting some suggestions, especially out of Asia, that that might not be the case. Given these concerns, how much weight should be put on the findings of the SIR models, Professor Woolhouse?

Professor Woolhouse: In the early stage of the epidemic—let us call it the first wave up to lockdown—things like herd immunity do not play a role. Too small a fraction of the population have been and are being exposed, nor have we reached anywhere near what we think might be the ceiling of susceptibility and the fraction that is acceptable[1], so it is not really playing a role.

In the longer term, the effect and duration of herd immunity will be very important, but, as I have already said, there are too many unknowns to make long-term predictions anyway, so I do not think those things had a big impact in the first wave.

[1] Note by witness: Professor Woolhouse said 'susceptible' not 'acceptable'.

Q804 **Aaron Bell:** You mentioned the first wave. You think we may have to live with Covid for some time. There has been a lot of talk about the risks of a second wave here and abroad, but there does not seem to be that much evidence of second waves around the world so far. With that in mind, how useful are models now going forward, particularly with respect to some of the policy decisions we are having to make about schools, quarantine and social distancing? How much weight would you put on models at this stage of the pandemic?

Professor Woolhouse: At this stage of the pandemic I would like to see the models used in a different way. The response to the epidemic in the UK should now be based on data—we have data from various sectors and sources—because, as Johan described earlier, this is a very complex non-linear system and it is hard to interpret those data. Therefore, the models will be very valuable indeed for helping us interpret it, but I think the response from now on has to be data driven.

Q805 **Aaron Bell:** May I put that to Professor Giesecke as well?

Professor Giesecke: I agree completely.

Q806 **Aaron Bell:** The original Imperial model that was credited with moving the Government to the lockdown position as rapidly as it did was a modified one drawn up for influenza. What specific limitations does that place on the model and any inferences we might draw from it?

Professor Giesecke: As far as we know, one major difference is that influenza to a large extent is spread by children, whereas for Covid this does not seem to be true. That is the big difference. We are also seeing more and more that Covid is not a homogeneous community spread and it is more a matter of clusters being infected, so it is a slightly different epidemiology from influenza.

Q807 **Aaron Bell:** Professor Woolhouse, you seem to agree.

Professor Woolhouse: I do. I would add that from a UK perspective the influenza models were working to a reasonable worst case for a possible influenza pandemic, which is a very sensible way to approach the problem. They have been doing that for some years, but it did become apparent fairly early on that the reasonable worst case for pandemic influenza did not match anywhere near perfectly a reasonable worst case for Covid-19, so inevitably there was a little lag while there was a readjustment for what a reasonable worst case might look like for this new disease.

Q808 **Mark Logan:** Professor Woolhouse, we know that three models coming from different universities supported the need for a full lockdown in early March. Do you agree with their conclusions?

Professor Woolhouse: That a full lockdown was needed? I do, given the situation we found ourselves in in March. I would characterise lockdown as a panic measure; it was something we did in the UK and was done around the world because we could not think of anything better to do

given the information we had available. I supported the lockdown at the time, but I was hoping very much it would be a short-term panic measure and we would move to more focused responses in the fairly near future.

Q809 **Mark Logan:** In your view, when did those models suggest we should go into full lockdown?

Professor Woolhouse: The timing of a lockdown is a very complicated affair, so I hope the Chair will forgive me if I try to give a full answer to this very important question. The circumstances we were in in March—my own group did some of the calculations for this for Scotland specifically—was that the epidemic was doubling in size every three or four days. That is very fast. That means even small delays are likely to have a very significant impact on the overall size of the epidemic, so it was important to move fast; there was not the luxury of long-term decision making.

With hindsight, when should the lockdown have been initiated? That is too simple a question. What I need to know as an epidemiologist and modeller is how hard the lockdown is. How long do you want it to be, and what are you going to do afterwards? If you can give me details of the planning that goes into the lockdown and post-lockdown stage, I can give you an answer on the best time to implement lockdown, but it is very simplistic to say "as soon as possible". You could make an argument for whether we should have locked down way back in February. I think that at the time you and most other people would have said that was a very disproportionate response given the situation at the time. Timing is a difficult thing in this sort of situation.

Q810 **Mark Logan:** With hindsight, do you think that perhaps different assumptions should have been used in the models?

Professor Woolhouse: I think the key driver, certainly from my point of view, of the decision to implement lockdown quickly was simply the doubling time. We were faced with an epidemic that was doubling in size every three or four days, and that tells me you have to do something and you have to do it quickly.

Q811 **Carol Monaghan:** Professor Woolhouse, if I can follow on from what Mark was asking, you said that all the models must be informed by data. The problem we have here is that human behaviour is part of the modelling—it can't just be about data. Is there a problem when looking at models that simply look at data, that we do not take into account what is actually happening or what humans will actually do?

Professor Woolhouse: The short answer to your question is, yes, it is a very big problem. I said right at the outset that I did not think we could make predictions for this disease. Behind that comment were two things I was concerned about. One was the role herd immunity will come to play in the long-term unfolding of this epidemic. We do not know enough about that, so that is one reason we cannot make predictions.

The other is exactly the one you said. We are being asked to predict how a human being will change both spontaneously in response to the presence of, let's face it, a very serious disease indeed and as a result of Government interventions and how they respond to regulations. I think that is very difficult to predict.

The models in play in March were, ironically, very strong on human behaviour, based on behavioural studies carried out—there are quite a number of large databases on this—that allow them to say who makes contact with whom and where the opportunities to spread an infection might lie, but all those databases went out of the window once lockdown happened.

Q812 **Carol Monaghan:** One of the things we kept hearing as parliamentarians was that we could not go into lockdown too quickly because people simply would not accept it for a long period. We now see that people have accepted it. Was a mistake made earlier in the assumption that people would not be happy to be locked up for weeks at a time?

Professor Woolhouse: I see Johan wants to come in on this point. I am going to answer your question by repeating what I said. I do not think we could predict that, so the right thing to do—my own group did this—was to predict a range of scenarios about different levels of compliance with the regulations, and we did that.

Professor Giesecke: I agree with exactly what Professor Woolhouse said. To model the change in human behaviour in the face of an epidemic is the most difficult bit of it because there is no way to predict that. That is true for Covid and AIDS and other diseases. Humans change their behaviour in the face of an epidemic.

There is an interesting example in Sweden. Sometimes we are blamed or are called names because we do not have a hard lockdown but a soft lockdown. In mid-March the Government said, "We have some restrictions we want to place on you. We will not impose them by law; there will not be policemen in the street, but we want you to diminish your social contacts." We can see now that the contact intensity in the Swedish population went down from 130 in a week, so it dropped by 70% just by the Government saying, "Please do like this."

Q813 **Carol Monaghan:** Referring again to the data, this afternoon we have already talked about the case fatality rate in terms of the modelling, but we have a problem because of low levels of testing throughout March, April and part of May. How do we get accurate models? We need to be looking at the infection fatality because we have no idea what that is, so how do we develop accurate models when we are looking at softening or coming out of lockdown?

Professor Woolhouse: You have again put your finger on a difficulty here. Most modellers would regard the data on deaths as the most reliable source of information on how this is spreading. To some degree,

we are going backwards with an assumed infection mortality rate to try to estimate the incidence of infection. That is definitely problematic.

The other problem with having to rely particularly on deaths is that they do not happen until typically a few weeks—say, three weeks—after the first infection. You are always three weeks behind what is actually going on at the time, so that is a big difficulty.

Q814 **Dawn Butler:** Professor Woolhouse, I want to pick up the last point in regard to the modelling using deaths as opposed to hospitalisation. Would it have been more accurate or beneficial if we had used hospitalisation as opposed to deaths?

Professor Woolhouse: I should not have given the impression that people are using only deaths. That is not correct. Hospitalisation and the results of testing are all included. Different models use different data streams in different ways. That is one of the reasons you are getting variations in, for example, the estimates of the R number: different models treat the data in a slightly different way. Whether you are weighting more to deaths or hospital cases makes a difference, but that is a very well understood and much discussed issue within the modelling community.

Q815 **Dawn Butler:** To drill down a little further, which is more useful to you, or how is it weighted?

Professor Woolhouse: Depending on how you choose to weight them—there is some subjectivity in this—you are likely to get different answers. My personal focus is less on largely asymptomatic or very mild cases in the community and more on locations where a severe outcome is very likely— care homes and hospitals being particularly important there. For that, you want models that look at the spread of the disease in those settings, not across the country as a whole. As I have said several times already, you need to be much more focused and targeted in where you are putting your effort.

Q816 **Dawn Butler:** My next question is to both professors. Evidence suggests that super-spreaders may account for a significant amount of transmission. To what extent was that modelled and identified?

Professor Woolhouse: Super-spreading basically means that some individuals for some reason pass on a lot more infection than others. It has a number of effects. One of them is that when the incidence of infection falls quite low we expect to see not a uniform spread within the community but clusters of infection. Indeed, some countries like South Korea seem to have been very successful in identifying clusters of infection—outbreaks, basically—investigating them and targeting their control efforts around them.

Again, it comes back to targeting. If you have evidence that there are super-spreading events, clusters or outbreaks, targeting and control

efforts around them gets you a lot further forward than just blanket measures across the entire community.

Professor Giesecke: From what I have seen—I am trying to remember—most models do not include super-spreaders. I think most epidemiologists agree that they form a rather small part of the overall spread; they are not very important epidemiologically for the entire pandemic.

Q817 **Dawn Butler:** I took the test today. One swab was taken of the back of the throat and the nose, but it counted as two tests. Originally, I understood that it counted as two tests because it was a test in two different places, but if it is using one swab why is it counted as two tests?

Professor Woolhouse: I cannot answer that, but I can give you a more general observation. I think the focus on simply the number of tests is extremely unhelpful, and setting targets based on numbers is quite unhelpful. What is crucial is a strategy to which the testing contributes. Why are you using these tests, and what is the goal of that strategy? Some strategies that might be very effective require relatively small amounts of testing and others require extremely large amounts of testing, so it is not the numbers of the tests that count.

Q818 **Dawn Butler:** Professor Woolhouse, at the very beginning you talked about communicating outcomes, which I think is absolutely vital. You talk about datasets and modelling and how you communicate with the general public. It is also vital in terms of how they respond when asked to participate in lockdown, for instance. You talked about communicating different outcomes. How do you compare what you think is the ideal way of communicating outcomes to the way we currently have—*[Inaudible]*

Chair: Professor Woodhouse, did you get the gist of that?

Professor Woolhouse: There was a break, but I think I did. I think the focus on the R number has been unhelpful in that regard. If I am a member of the public and want to assess my risk, I do not think the R number helps me do that in any shape or form; it certainly does not help me personally.

The other focus is on the incidence—the number of new infections a day. That is a bit more helpful perhaps, but it probably needs to be more granular than we are able to provide at the moment.

The thing I would really want to know is my risk. What is the risk of my becoming infected? What is the risk of my becoming diseased and ill? What is the risk of my going to hospital, and what is the risk of dying? That is the sort of thing we should be communicating, and I do not believe that as part of the scientific community we have succeeded very well in communicating where the risks really lie. As I have said several times, they are vastly disproportionate in the elderly and frail. That needs to be very clear; everyone needs to understand that.

Professor Giesecke: Maybe we have been there even in Sweden. I do not think many people are aware of the tenthousandfold difference that has been mentioned, so that is one example.

Q819 **Dawn Butler:** But Sweden has the highest death rate per million people.

Professor Giesecke: We are up there with the UK; we are not that different from the UK.

Professor Woolhouse: The epidemic curve for Sweden overlaid on that for Scotland is indistinguishable.

Q820 **Chair:** That is very interesting. We might come back to that point.

Professor Woolhouse, on the comment you made about the R number, as a member of SPI-M concerned with modelling, has the R number been the key focal point in those discussions and the advice you have sent up to SAGE, or has it taken on a prominence either in SAGE or when received by the Government? How has it acquired this prominence in SAGE?

Professor Woolhouse: I am sure my colleagues in the next session will also have a lot to say about that. I think the general feeling in SPI-M is that perhaps we have created a monster. We have got attention on R, which was the right thing to do because it is an extremely important epidemic parameter, but now very close attention is paid to it and less attention is paid to the other measures I referred to in my answer to Dawn Butler that I think are more important, certainly for the general public, in understanding what is going on.

Q821 **Chair:** Risk being one.

Professor Woolhouse: Risk being at the forefront of that.

Q822 **Graham Stringer:** Professor Woolhouse, may I take you back to Mark Logan's questions and your answers about the doubling time being about three days and the importance of that in the timing of the decision to go to lockdown? Was part of the problem that the model stayed with the doubling time from the original data from Wuhan and did not look at the doubling time within the United Kingdom—it stayed with the old data too long?

Professor Woolhouse: I do not think that would be a fair assessment. I think that as the epidemic curve progressed it became obvious what the doubling time was because we were measuring it. Remember, we are talking about a very short period between the first week and last week of March. As the data became apparent, as you saw, there was a rapid reappraisal and a Government response accordingly.

Q823 **Graham Stringer:** I read the minutes of the House of Lords Science and Technology Committee, which state that Ministers were using the doubling time from Wuhan when the statistics in this country were, as you say, three or four days. That was why I asked the question. Do you

think that that evidence to the Science and Technology Committee was unfair?

Professor Woolhouse: I did not see that evidence. For the model projections, yes, it is true they were parameterised from Wuhan because, like anywhere else in the world, that was all the data we had, but my own group's work was calculating the direct doubling time and what we were seeing, not projections from Wuhan or anywhere else.

Q824 **Graham Stringer:** May I ask you about granularity and the problems it causes in the models, particularly the fact that there does not seem to be a distinction between infection that takes place out of doors and indoors, and whether that has any implications for the policy and the timing of coming out of lockdown?

Professor Woolhouse: I think it does. The important thing to realise here is what the models require as a contact: somebody meeting somebody else with a chance of passing on the infection and how they define that. But that is done just on a population level basis; it is not done with granularity—for example, whether that contact was indoors or outdoors. Was that contact in a non-essential retail outlet, which is another policy decision under debate, or what might be the role of extra desk spaces in schools? The models do not have that granularity, so when Government is asking questions like that we are starting to run out of road in providing model-based answers.

As I keep coming back to, we have to go back to the evidence and data on those kinds of interventions. The difficulty for everyone in the world, not just the UK, is that this is a new virus and we do not have the evidence base to act on. We are having to respond very quickly to evidence that changes, accumulates and sometimes contradicts itself, and we have to make judgments on that. It is not easy for anybody. The models do not have the granularity needed to make these very fine judgments.

Q825 **Graham Stringer:** You said the R number had probably been overused. In the north-west at the present time one of the six models has said that the R number has just crept marginally over one, and that is being used as an argument to slow down coming out of lockdown, particularly in relationship to schools. Rather than the R number, is not the more important information where the infections are taking place? If most of the infections are taking place in hospitals and care homes, the R number is more or less irrelevant, is it not?

Professor Woolhouse: I would never call it completely irrelevant—I am an epidemiologist—but I very much take your point that R does not tell you where the infections are occurring. As I have said many times previously, a few weeks ago our estimate was that R was below one in the community but greater than one in care homes. Quite frankly, if I had to take one or the other, I would like it the other way round. The last

place we want this infection spreading like wild fire is in care homes, so it detracts focus from what really matters.

Q826 **Katherine Fletcher:** As is often the case when following such an august member of the Committee, Mr Stringer has slightly stolen my thunder.

I have a background in biology. I have gone through the submissions for the next panel. One striking thing is the granularity that the models offer versus the specificity we have been giving in the lockdown rules. For example, in my admittedly amateurish assessment it does not seem that we have any capacity to understand care homes versus families of six going to a shop versus the risk of giving your mum a quick hug. It seems to be in a block of 15 minutes within 2 metres or nothing.

Professor Giesecke, would you suggest it is possible to do modelling and we have just run out of time because this is new, or is the level of complexity I am articulating not possible?

Professor Giesecke: It is difficult to say the least. There is a growing awareness that this disease is clustered, compared with influenza, which is commonly spread in the population. That makes what you are asking for even more difficult. One thing we should do in both countries is put more emphasis on where the spread of infection is taking place and doing that by testing and contact tracing, trying to find out which places are the hotspots for the spread. In my country at least, we are not doing that very much yet. Is that an answer to your question?

Q827 **Katherine Fletcher:** Most certainly. I am trying to understand whether we have missed a trick in the way we have structured the models. Rather than going for that very generic influenza-based model, perhaps we could have done something that was more about what happens if something occurs in this setting. Professor Woolhouse, you have mentioned developing models targeting care homes, et cetera. What, if any, capacity could we have had to do that earlier?

Professor Woolhouse: We are developing that capacity now, and my colleagues in the next session can probably give you more chapter and verse on that. This partly comes down to our readiness in the UK, across Europe and North America, being directed at pandemic influenza. Remember, these models are originally influenza models and are obviously appropriate in that context, so they concentrate on things like schools. Schoolchildren are very important in spreading influenza and they are very susceptible to getting it, sometimes quite severely, so that is important. Therefore, they did not have care homes in them because they are influenza models. Obviously, care homes are important in that context, but the focus was on schools.

That is not the appropriate focus for this new disease. We were not missing a trick; we were preparing our capacity, including our modelling capacity but in many other ways, for pandemic influenza.

I am very much interested in Johan's view on this. There is a view that in general the countries that experienced the SARS epidemic in 2003, particularly in south-east Asia—never mind the modelling—were better prepared to deal with this infection, which is much more like SARS in some respects, whereas the countries preparing for pandemic influenza had to recalibrate a little as this new infection, which was not the one we were prepared for, hit us. Do you agree with that, Johan?

Professor Giesecke: I agree with that. There is a clear difference in the way preparations have been made in south-east Asia compared with Europe.

Chair: We thank both witnesses very much. As has been evident through this session, at the beginning of an outbreak, as Professor Woolhouse said, there is a need for emergency measures, and pressing the emergency stop button, if I can put it that way, is perhaps the only thing that is open to Governments around the world, but the benefit of time passing and the experience of countries around the world, including our own, is that one can make more finely calibrated judgments. I think the evidence we have heard from both witnesses today has shown in different ways—whether it is the number of factors in addition to epidemiological modelling and some of the inputs into those models—that this is an opportunity for us to take advantage of that learning. We have been able to have expert evidence to that effect. We are very grateful for it.

It is very important as we draw lessons not just in the long term about how this pandemic has been handled, but we are very keen to be able to make recommendations to Government, as we already have, on lessons that can be learned and applied now for future steps. Thank you very much indeed for your evidence today.

Examination of witnesses

Witnesses: Professor Ferguson, Professor Keeling and Dr Davies.

Q828 **Chair:** The Committee is very pleased to welcome Professor Neil Ferguson, Professor of Mathematical Biology at Imperial College London, who has appeared before in this Committee; Professor Matt Keeling, Professor of Mathematics and Life Sciences at the University of Warwick, who is a member of the Scientific Pandemic Group on Modelling, SPI-M, which we heard about in the last session; and Dr Nicholas Davies, a research fellow in mathematical modelling at the London School of Hygiene and Tropical Medicine, who is also a member of SPI-M. Thank you very much indeed for giving us your time today.

I shall kick off with some questions for Professor Ferguson. Thank you very much for coming back to the Committee; your evidence is very much appreciated. You no longer attend SAGE, but do you continue to advise the decisions taken by Government?

Professor Ferguson: I do. SPI-M does not have a fixed membership. I attend SPI-M meetings and other advisory meetings on an ad hoc basis.

Q829 **Chair:** Does the model that you have worked closely on with your team, and which has informed decisions, continue to be available?

Professor Ferguson: That continues to be available, as do a number of other models.

Q830 **Chair:** When you last came before the Committee, on 25 March, you estimated that UK deaths from the coronavirus would be unlikely to exceed 20,000 and could be much lower. As we know, the official death toll is over 40,000. What went wrong?

Professor Ferguson: I think there are two things. There is a paper out in *Nature Today* that highlights that, around about that time, just before lockdown in the first two weeks of March, we probably had 1,500 to 2,000 infections imported from Italy and Spain. We just had not seen any surveillance data until that point—so there was much heavier seeding than we expected.

To go back to our report 9, we looked at a range of scenarios. The key thing with the number of deaths is at what point in your local epidemic you trigger interventions and how far in you are when you shut down transmission. Frankly, we had underestimated how far into the epidemic this country was. That is partly the reason.

The second part, which I think would have been more avoidable, is that half of those deaths occurred in care homes. We have always worked under the assumption, which was Government policy at the time, that care homes would be shielded from infection. What we have actually seen is infection rates that are probably four times higher than in the general population, in care homes, with a very vulnerable population group. That death toll would probably have been about half of what it is now, had we had—

Q831 **Chair:** Your sound has broken up, Professor Ferguson. Did I hear you correctly as saying that the death toll would have been half what it has turned out to be in care homes, if what had happened?

Professor Ferguson: Not in care homes, but overall. Roughly half the deaths we think are related to care homes. That is not a controversial statement. The ONS came to a similar conclusion earlier this week.

Q832 **Chair:** Indeed, absolutely.

On the seeding, at the time the forecasts were made at the beginning of March, you had reason to believe that there were fewer cases in the country than there were at the time that had come from Italy.

Professor Ferguson: Yes. We tried very hard to estimate what proportion of cases were being missed. Obviously, at the time we had a policy of trying to screen people at borders, and we estimated then that

maybe two thirds of imported cases had been missed. What we now know, because the epidemic took off in Italy and Spain before anybody had realised, is that probably 90% of cases imported into this country were missed by those border measures, because we were not checking people.

Q833 **Chair:** That was my next question—that we were not monitoring who was arriving with the disease.

Professor Ferguson: Sorry, could you say that again?

Chair: The reason we missed them was that we were not making checks at borders to be able to detect people with Covid. Is that right?

Professor Ferguson: Not from those countries.

Q834 **Chair:** You were a member of SAGE at that time. Did SAGE advise that we should be making tests, not least to detect whether it was an important source of seeding?

Professor Ferguson: This is really about decisions by the Foreign Office and the Department of Health and Social Care, not by SAGE. SAGE recommended that where a country had been identified as having active transmission we should check travellers from those countries. The difficulty was that, as we now know, Spain and Italy, which was the source of many infections into the UK, had large epidemics before they even realised. So we were just not aware of the scale of transmission in Europe as a whole.

Q835 **Chair:** Nevertheless, looking back, given that we underestimated the number of seeded infections that we had, notwithstanding those difficulties, would it have been a better precautionary move to have tested people coming in from all countries, or some countries?

Professor Ferguson: Had we had the testing capacity—and we have to bear in mind that there were testing capacity limits—certainly screening everybody with symptoms coming in would have given us a much better impression of where infection was coming from.

Q836 **Chair:** I see. So, again, it was to do with the capacity, rather than a view that—

Professor Ferguson: It was to do with testing capacity and PHE capacity to actually implement that on the huge numbers of travellers coming in from Heathrow and other airports.

Q837 **Chair:** I see. The next element that you mentioned was care homes. In building the model and making the projections you did from that, why was the role of care homes and infections in care homes under-predicted? Did it have too little weight in the model, or was the experience in care homes different from what the model had assumed, in a way that it was not possible to anticipate?

Professor Ferguson: I should say, first—and I listened to some of the last session—that the model that we used is not specifically a flu model, or anything else. It has a very flexible representation of different types of places. We are now integrating care homes into that. At the time, we did not have enough data really to understand what levels of transmission we would see in care homes.

We also made the rather optimistic assumption that somehow, as was policy, the elderly would be shielded, and particularly the most vulnerable would be shielded, as the top priority, and that simply failed to happen.

Q838 **Chair:** During that time, SAGE was meeting twice a week. Obviously, you were working on the modelling. Was the experience of what was happening in the real world, if I can put it that way, being detected, reported on and fed into SAGE, so it could then be fed into the modelling?

Professor Ferguson: I should say at the outset that we do not just sit in front of computers and build models and code. Most of what we do is to analyse the data streams available to us, and that has always been the case, going back to early January. Our earliest work was just to look at the data, but the data has grown over time and we now know a lot more about this virus than we did back in March, and certainly more than we did back in February and January.

Q839 **Chair:** When did SAGE begin to be aware and attach importance to the experience in care homes?

Professor Ferguson: We anticipated in theory the risks to care home populations—I think, back in meetings in February and certainly early March; that was discussed in SAGE meetings at that point. Graham Medley, the chair of SPI-M, myself and John Edmunds all highlighted the fact that here was an exposed population that could be at a very significant risk. I think that both the CMO and Patrick Vallance also completely recognised that.

Again, issues were raised about our ability to do testing. The only way in which you can really protect care homes is through extensive testing to make sure that infection does not get in. Concerns were raised about the ability to do that at the time.

Q840 **Chair:** But at that time, when you came before the Committee on 25 March, the projection of deaths being unlikely to exceed 20,000 was based on the policies that had been taken on the lockdown, with visibility of the policy suite across the board. You knew what the policy was—SAGE knew what the policy was, for care homes—so why was the experience of care homes beyond what you knew at the time?

Professor Ferguson: The policy has always been to protect care homes and the elderly. The policy has always been clear in that sense. This is not unique to this country; the policy has simply failed to be enacted until very recently, and there are multiple causes of that.

We understand a lot more about the transmission dynamics occurring in that sector. One thing that we did not anticipate at all at the time was that, in the larger care homes, it is very common for staff to work in more than one facility. That accelerated the spread of infection from one care home to another. We have learned a lot more since then. At the time, we were not anticipating the epidemic in care homes to be close to the size it turned out to be.

Q841 **Chair:** The third leg of the explanation that you gave was that we were further down the path of the pandemic than was supposed at that time, towards the end of March. Can you expand a little on that?

Professor Ferguson: That is just a reflection of the first thing. The higher level of seeding of infection in the country meant that, effectively, at the end of March—bearing in mind that we did not see the epidemic peak until mid-April, in terms of hospitalisations—there were a lot more infected people around than we had anticipated.

Q842 **Chair:** So, knowing what we do, and having acquired more insights—and at the end of the last session you will have heard reflections that you gain insights and evidence as you go through—what is the current prediction of the number of fatalities that we will experience in the UK?

Professor Ferguson: It slightly depends on which fatalities you are talking about. If we talk about what the ONS reports as Covid-related deaths, the death numbers are going down quite quickly. I do not have a precise number to hand, but I suspect that it will be of the order of 50,000 or so. That is not a precise prediction. You will be aware that we are working on the computation of excess deaths, and we will be reporting on that shortly. If you look at overall deaths, you get a higher figure from the net impact of the epidemic.

Q843 **Chair:** But at the end of March, on 25 March, you told the Committee that the number of deaths was unlikely to exceed 20,000 and could be much lower. What is the like-for-like current comparison? What do we now project on that same basis?

Professor Ferguson: The challenge is now what you assume about the effect of relaxing lockdown. Do we see an increase in transmission, or will there be a second wave? If you are talking just about first-wave deaths, depending on precisely how you count them, I would say around 50,000. I do not have the numbers in front of me, so I cannot give you a range around that, but that is the sort of figure. My colleagues on the panel may be able to comment more.

Q844 **Chair:** Thank you—we will come on to that. One reason for the Committee's inquiry is to be able to learn lessons on the way. Some of the learning is to look back on when the pandemic has concluded but, sometimes, decisions will be repeated. Coming out of lockdown may have certain commonalities with going into lockdown. That is why I want to ask for your reflections, looking back. Is it your view that the right decisions were taken at the right time?

Professor Ferguson: I think that the right decisions were taken. In retrospect, as my colleague John Edmunds has said, and I completely agree, had we introduced those measures—

Q845 **Chair:** Could you pause for a second? We have some very bad feedback and interference.

Professor Ferguson: The epidemic was doubling every three to four days before lockdown interventions were introduced, so, had we introduced lockdown measures a week earlier, we would have reduced the final death toll by at least a half. So, while I think the measures were warranted, given what we knew then about this virus in terms of its transmission and locality—and I would not second-guess them at this point—certainly had we introduced them earlier we would have seen many fewer deaths.

Q846 **Chair:** Looking back now, with the great benefit of hindsight, were there any other lessons that it is possible to identify, which may have some relevance for decisions coming up?

Professor Ferguson: I would completely agree with Johan Giesecke and Mark Woolhouse that a focus on where transmission is happening is absolutely critical to coming out of lockdown. We want to be in a position where we can identify clusters of infections very rapidly and impose locally targeted interventions in a way that are not as economically destructive as overall lockdown measures. For multiple reasons, we were not in a position to be able to do that in March, with the testing or the understanding of transmission, but we are on a better basis to do it now.

Q847 **Chair:** Before I come to my colleagues, perhaps I may ask that question of Professor Keeling and Dr Davies. Looking back at this point, are there any decisions that it is clear could have been made differently and it would have been better to make differently, which we ought to be aware of for decisions coming up?

Professor Keeling: Hindsight is a wonderful thing when it comes to looking back at epidemics. It is always easier to say that we could have done something slightly different. I would echo Neil's comments: that with hindsight we could have gone into lockdown earlier. One of the main constraints that we were facing at the time was the advice that the population at large would resent a very long lockdown, so we were almost balancing that against the chaos that a lockdown would cause—and, in the very early stages, we did not quite know. We knew that it was doubling every three to four days, but the data was still quite sketchy, certainly on how much it was going to double in the early phases in the UK. Lockdown more than a week before we did would have been very difficult to put through.

In hindsight—yes. One question in looking forward is where these outbreaks are going to occur in future. We are not going to see a uniform spread of infection across the country. It is going to be very isolated in small pockets. It is really going to be about how we target those with the

test and trace scheme and make sure that that information flows readily throughout PHE and the control zones.

Dr Davies: I completely agree with what my colleagues have stated so far. It is clear from our own modelling looking back that an earlier lockdown would have been substantially better in terms of the health outcomes that we see, in terms of deaths. Moving forward, having a better situational awareness of locations of infection and where it is spreading will be increasingly important.

Q848 **Graham Stringer:** It is good of you to spend so much time with the Committee, Professor Ferguson. The last time you were here, you told us that we did not know whether the British summer was going to reduce the infection rate of the disease. Do we now know?

Professor Ferguson: There have been some studies, though not done by ourselves. A group at Harvard and a couple of other groups have looked at the effect of climate on coronavirus transmission. They conclude that there might be evidence of perhaps a 10% drop in R—let us call it R—20% from the worst time of year, which is November, and the best time of year. That is a 20% difference in R, roughly. So there may be a small effect, but it is not dramatic.

Graham Stringer: Is the sound system working?

Chair: Yes, I think it is. Carry on.

Q849 **Graham Stringer:** To go back to what you were saying about protecting residents in care homes, when NHS England decided to send people back to care homes out of hospitals to create capacity for intensive care, was there not concern within SAGE that this was going to intensify the epidemic?

Professor Ferguson: To be honest, I do not remember that being discussed in SAGE. I may be mistaken but, off the top of my head, I became aware of it only later.

Q850 **Graham Stringer:** When you present papers to SAGE—and I realise that you are doing most of this work at pace—what quality assurance do you have of those papers before they are presented to SAGE?

Professor Ferguson: That is perhaps the most important point. Things were being done at 2 am. Because of the pace involved, the quality assurance is achieved by comparing results arrived at independently by different groups through SPI-M—so SPI-M acts as a very rapid peer-review and model comparison mechanism. We have learned from many other infectious disease crises that the best way to test any one model is by comparing it with a range of different models. If they agree, you have some confidence, and, if they disagree, you get some better insight into uncertainty in underlying data or assumptions or into potential problems with individual models.

Q851 **Graham Stringer:** I do not know whether you heard the previous discussion, but they talked about the reliance on models and the information that goes into those models. Do you think that the Government and SAGE have been over-reliant on models? I know that you are a modeller.

Professor Ferguson: Yes, you are asking me—a turkey voting for Christmas.

Graham Stringer: Yes, I understand that.

Professor Ferguson: I think that Sir Patrick and Chris Whitty have a healthy scepticism for models, and the clinical and virology communities were both represented on SAGE. You could argue about the balance of representation. What models do is to codify assumptions and knowledge in a very precisely testable way, so I would defend them from that point of view. Where they become problematic is if they are taken as in some sense a literal view of the truth. Models can only be as reliable as the data that is feeding into them.

Q852 **Graham Stringer:** I have one final question. The Canadian academic Meunier has said that the lockdown has not affected the death rate or fatality rate at all, and the curves were going in the direction that they followed at the time the lockdown happened. What is your response to his views?

Professor Ferguson: I think that there is overwhelming evidence from large numbers of academic groups and large numbers of countries about the mortality impact of this pandemic. We have seen huge spikes in all-cause mortality, unheard of in the last 20 to 30 years, particularly in the most heavily affected countries, like the United Kingdom, but across the European countries. We have seen the same unfolding in Brazil today, and in New York State, so there is overwhelming evidence against that hypothesis.

Q853 **Andrew Griffith:** I shall carry on, if I may, where Graham left off. If I may say so, you seem much more abashed today in giving evidence to the Committee than when we met in March.

Professor Ferguson: I do not know why that might be.

Q854 **Andrew Griffith:** I want to prosecute the point a little bit about the reliance on the model. As you say, Professor Ferguson, they are not single-point versions; the model has a set of assumptions nested in it, and often the best way in which to convey a range of outcomes is through the sensitivities. You said yourself that, at the critical moment at which decisions were being made in real time, cases were doubling every two to three days. Do you think that the model successfully articulated that point? Were you presenting multiple scenarios, for example, or was there a danger that people themselves alighted on a single case and became over-vested in that?

Professor Ferguson: I will let the others speak for themselves, but we always look at a range of intervention scenarios in terms of the interventions being modelled and the timing of their introduction or thresholds that would be governing their introduction.

There was lots more sensitivity analysis that could have been done, and we just did not have time, given the pace at which we were working. I think that there was a good appreciation, maybe not quite of the pace of doubling: our estimate is that it was about 3.3 days at the peak, at the fastest-moving time and, at the time, we thought that it was more like 4.3 days. So that was out. But, still, a doubling even of every four days is clearly something that requires rapid action, and that was understood.

Dr Davies: In London School's models, our central estimate was also around a 4.3-day doubling time, but we included substantial uncertainty around that value. We derived our values for doubling time via measures of R0 in settings without substantial control measures in place. A variety of studies—I think, 11 overall—showed that there was substantial uncertainty in the doubling time.

Throughout with our models, we presented the uncertain outcomes that stemmed from that uncertainty over doubling time, as well as summarising a number of different potential scenarios of timing and adherence to non-pharmaceutical interventions. While it looks like the doubling time that we actually saw in the UK was at the higher end of the estimates that we were producing, you cannot always get the central estimate bang on, and we followed appropriate steps to communicate the uncertainty around that central estimate.

Q855 **Andrew Griffith:** Do you not think that a central estimate in itself, given how many unknown variables there were at the time, allowed a sense of false certainty to be propagated?

Dr Davies: That is a difficult question to answer. I do not think that it is very common not to provide a central estimate—you need to anchor the estimates somewhere.

Professor Ferguson: Can I make an additional point around the difference between central estimates and reasonable worst cases? Throughout February and March, SAGE was planning against a reasonable worst-case scenario, particularly in terms of severity.

Q856 **Andrew Griffith:** We have a set of models. What was the process subsequent to that? What is the period when they are formally reissued? Is it once a week, or is there a formal release as new data moves very rapidly over time? To bring us bang up to date, when was the last time you issued or released a model?

Professor Ferguson: I am not sure that it works like that. These models are under continuous development. One of the benefits of things like GitHub is that you can always roll back. We are constantly looking at new things with a variety of models, not just one model.

The way SAGE and SPI-M operates is that, while there are certain routine things we do—such as produce the weekly forecasts and weekly estimates of R—most current activity is more task based. SAGE or Government will have a—*[Inaudible]*—it may be better to look at transmission in the next month. Groups contributing to SPI-M will all independently go away and try to answer those questions and come back often two days or three days later, after working all night, with our best estimate of the answer; then those will be compared.

Q857 **Aaron Bell:** I do not want to speak with the benefit of too much hindsight, but I want to follow up on what the Chair was saying earlier. I understand that the degree of seeding seems to have been the biggest reason all the models may have underestimated the total number of deaths so far in the UK. However, given the growing understanding regarding transmission, and the fact that a lot of these models were based on influenza transmission and more on transmission on to children, and so on—and this might not be the case for each of your models, so I shall ask you all individually—do you stand by the conclusions and recommendations that you made in the early stages, on the basis of what you knew at the time?

Professor Ferguson: Yes, I do. Given what we knew at the time, there was no other option if we were going to avoid very large numbers of deaths.

Professor Keeling: First, I would like to correct the assumption that all the models are based on flu. Ours was set up and is bespoke to look at Covid. We have flu models and, obviously, we use that experience in generating new models, but this was all based on what was known from Wuhan. We were very focused on the fact that this had a lot of age structure that was very biased towards the older age groups.

As for standing by our initial conclusions, we were also making forecasts that went to SPI-M and then were taken to SAGE in early March. At the time, with an uncontrolled outbreak, it was maybe up to 500,000 deaths. Actually, even with the best parameterisation that we have now, we are not too far off that scenario, if we went back and reran it from the beginning. So it is very obvious that we needed a lockdown very early on.

Q858 **Aaron Bell:** To give you a chance to speak further to what you have just said about your model, does that mean that your model took into account settings like care homes in the way an influenza model would take into account settings like schools, or is it an age stratification only?

Professor Keeling: Unfortunately, it was an age stratification only. We knew how important older people were, but at the time—certainly very early on—there was very limited data on care homes. I remember asking at some point, probably late March, what we knew about care homes, and we did not even know how many people were in care homes at that point. We can only generate models from the data that was available, so, to a large extent, we should have realised that care homes were probably

more important and put a higher focus on keeping asking about data and looking at infections there.

Dr Davies: The model that we developed at the London School for Covid was a bespoke model developed to match features to recapitulate the epidemics that we had seen from Covid. It includes a strong age-stratified component whereby we assume that children are less infectious than older individuals.

In spite of the fact that we developed it basically from scratch, we ended up with a model structure that is somewhat similar to the model structure that you would use to model influenza, simply because they are both respiratory pathogens. I want to push back a little bit against the idea that it is completely inappropriate to start from influenza as a model. In particular, we ended up with a very similar structure to what Professor Ferguson ended up using in his modified model.

Q859 **Aaron Bell:** Again, with a little bit of hindsight—and this speaks to a point that my colleague, Carol Monaghan, made in our first panel—there seemed to be an overall assumption that lockdown was sustainable only for a certain period of time. That is obviously true in the abstract, but it seemed to be very set about how long that time should be, and the assumption was made then. Would you have changed your recommendations if you had had a better handle on that aspect of human behaviour?

Professor Keeling, based on what we have seen, how does that relate to your model's idea of temporarily relaxing and reinforcing lockdowns, and what it would do about adherence to lockdowns?

Professor Keeling: The early assumption was that lockdown probably was not sustainable for more than four to five weeks, and we have already seen that that is blatantly not true. We vastly underestimated the general public and how reasonable they can be in the face of such an outbreak.

What it tells you about moving forwards is more difficult. We have always known that predicting human behaviour is very hard. Viruses are quite simple and do quite simple things; humans are much more complicated, and we interact with each of them in very non-linear ways. So trying to predict how humans would respond to a change in policy whereby we are locked down for so many weeks and then released again is very difficult.

We were just putting it out as a potential way, if we came out of lockdown too fast, of doing something regionally that at least enabled the NHS to carry on, in a sort of "save the NHS"-type framework.

Dr Davies: I would not agree that our lockdown model specifically assumed a four to five-week period that was sustainable. I am looking at my own paper, and the lockdown periods last anywhere from two to four months. I do not think that it was really a strong assumption going in.

Professor Ferguson: We also did not assume a particular duration for lockdown. Our report 9 was looking at lockdown periods of three months and policies after that, as necessary. There was no predetermined duration.

In the work we have been doing since, we have all been very focused on trying to understand what can be done short of lockdown—if we get clusters of transmission resurgence, how contact tracing, cluster detection and local measures can give us the same effect in controlling transmission, without the economic cost of lockdown.

Professor Keeling: Sorry, I was not saying that we were only thinking of a four-week lockdown, but in the early stages there was this assumption that lockdowns would probably have to be short, and that may be one of the reasons we did not go into lockdown quite as soon as we could have done.

Q860 **Katherine Fletcher:** Professor Keeling and Dr Davies, thank you both for your comprehensive and well-read papers. I know that you both submitted them on a Sunday, and it is genuinely appreciated.

The prize for the best comment goes to Professor Keeling for saying, "That question doesn't make any sense." Let us try to shed some light on it for the benefit not only of the Committee but of the general public watching.

I note lots of references to scientific phraseology—things like "intensive non-pharmaceutical interventions are recommended." For the British public, that means lockdown measures, does it not, to stop the spread of the Covid disease? Is that correct?

Professor Keeling: It could be lockdown and intensive social distancing, but it could be other things. It could be mask wearing, and we should not forget hand hygiene in all this. All these are important non-pharmaceutical measures.

Q861 **Katherine Fletcher:** I tried to understand the modelling that led to the different types of lockdown measures—the non-pharmaceutical interventions. I could not help but notice that lots of the models are based on a threshold from survey data, modelling how people interact with each other, and the interactions from those self-reported surveys have then been categorised to meet the "within 2 metres for more than 15 minutes" criteria, which seem to be a baseline. What I cannot see is how that feeds into the models or whether there has been any critical evaluation of that. Can I start with Professor Keeling?

Professor Keeling: That is a really good question. One of the biggest problems that we have with all this modelling—and I was listening to the questions in the session before, when you asked about granularity—is how you translate the risk of any particular action into a risk of catching infection, such as what the risk is of reopening small shops or someone

going to the hairdressers. Translating that into a model input is very difficult.

For most of what we have done, we have taken an initial estimate based on these surveys that we have, so we know where and when people meet and what types of interaction they have. We do not tend to just do this cut-off of 2 metres and a given time; we look at how long the interaction is as well. Obviously, longer interactions that are much closer are at a far higher risk. We use that as an initial calibration of the model fit. After that, a lot of what we do is just matching to the available data.

We knew that early on, once lockdown came in, we were going to have a decline in cases, but we could not have put hand on heart and given you an exact number on that. We know that a certain proportion of interactions will have stopped, but that does not correspond to exactly the same proportional reduction in transmission, because some of the interactions that happen just will happen, and they are maybe the stronger ones that did more of the interacting.

A lot of it is matching to the available data that is coming out. As Neil said, it is not so much about inputs going in; it is also about using these models to interpret the data that is around, so they are more of a statistical tool.

Q862 **Katherine Fletcher:** I was going to come to that later, but since you have mentioned it I will pick it up. I think in your submission, Dr Davies, you talk about being able to "fit our models to the outcomes observed in the UK." What is it that you had to change to make the models fit the observed outcomes?

Dr Davies: This goes through a formal statistical process, where you fit a number of different variables that go into the modelling; the statistical process then does imprints over those variables to see which values are consistent with the data.

Q863 **Katherine Fletcher:** I noticed an example from the internet.

Dr Davies: Right, okay. Some of the variables are very boring things—well, not necessarily boring, but things like the date of introduction of the virus into the country and the R0 at which it initially spreads. Some of them are a bit trickier to fit, such as how exactly rates of contact between individuals change once lockdown is introduced. Different groups use different approaches for that; we have been trying to use Google mobility data, which tracks people's aggregated movements into different categories of locations—workplaces, parks, residences and so on—and tries to link those to the observed patterns. The biggest discrepancy that we have seen arises from that fitting process.

Another set of data that we fit are more epidemiological parameters, such as the case fatality ratio and the hospitalisation rate.

Q864 **Katherine Fletcher:** Both of which have had to go down a touch from

the initial inputs.

Dr Davies: The hospitalisation rate has gone down, yes. In our case, the case fatality ratio is about right, or maybe slightly low. It depends on which dataset of deaths is being referred to.

Q865 **Katherine Fletcher:** One thing that struck me, if I was trying to explain this to a lay person, was how we get from the models to the point that Professor Keeling has just referenced, of what investigation has been done into the evidence on those briefer interactions. I am sure that a lot of members of the Committee will have a variant of this question in their inbox: "Why can I go to work, but I can't give my mum a quick hug?" because it is not spending 15 minutes under 2 metres. It does not seem to me that the models can talk to those types of questions. Is that true?

Professor Keeling: I think that is very true. We cannot categorise. With giving your mum a quick hug we do not know the risk factor. The question is whether you want to put your mum at greater risk—I presume that your mum is more elderly, and, as Mark was saying, older people are at much higher risk.

There is also the risk of asymptomatic transmission, which we do not know. In general, we want people to err on the side of caution. If you go back to work, hopefully you can try to keep 2 metres' distance from individuals—and 2 metres and 15 minutes are just a guideline; they are what we use in a lot of cases, but they are not absolutes. Obviously, 14.5 minutes is still risky and 16 minutes is not as bad as being with someone for two hours. It is a gradual thing. The whole idea is to be aware—to use the latest phrase—of the risks that you are taking all the time.

Q866 **Katherine Fletcher:** To play that back to you, you are saying that none of that is modelled; the models are at a level of detail way above that.

Professor Keeling: They have to be at a level of detail above that, because we just do not know what people do on a daily basis. We cannot forecast to that level of granularity.

Dr Davies: I do not have too much to add to that, except to say that one benefit of using these slightly more high-level measures of contacts, as in the contact surveys, is that it is easier then to follow up on and measure in real time how the pandemic has affected those measures of personal contact.

One project in the London School is ongoing telephone surveys of people's behaviour, which we can compare directly to earlier datasets, which have been collected to get an approximate understanding of how those patterns have changed over time. So it is not just about the level of detail in modelling—it is also about the level of detail in data collection, which might be prohibitive for the level of specificity you are talking about.

Q867 **Katherine Fletcher:** So it is rate of change that you are assessing, as

opposed to granularity.

Dr Davies: Somewhat, yes.

Professor Ferguson: I was just going to make the general point that it is not—

Katherine Fletcher: Oh, I think we have broken you.

Professor Ferguson: —very much in line with what my colleague said.

Chair: Will you start your answer again? We missed the beginning of it.

Professor Ferguson: We model aggregate contacts. The key concept of getting R below 1 is that of a contact budget. If we want to maintain control of transmission, only a certain number of contacts can be permitted; then it is a policy maker's decision, if they want to keep R below 1, which types of contact to prioritise. You might prioritise key workers and certain leisure activities, but that is not for us to decide.

Q868 **Katherine Fletcher:** No, but it is for the Committee to understand the response, and this is valuable evidence towards it.

I noted within Professor Keeling's paper an interesting reference, where it said that, if you use that baseline of within 2 metres for 15 minutes or more, it will allow 80% of cases to get caught with test and trace, which is being set up, but that there is an average of 36.1 individuals who need tracing.

Given that Professor Ferguson has just introduced the concept of a safe number of contacts, and the evidence that we heard from the first panel about giving members of the general public an ability to assess their own risk, could you speculate or give us a number that your models allow for any given person to help us to keep control of this virus?

Professor Keeling: We did the paper you are talking about in the very early stages, before we got many cases. It was about contact tracing pre-lockdown, which is why the numbers are as big as 36 per person. We were talking about throwing everything at the contact tracing to try to eliminate individual cases coming in.

Trying to give someone a figure for how many people you could meet to keep R below 1 is incredibly difficult. As Mark Woolhouse was saying in the first session, it very much depends on the age of that individual, as well as all the other factors that we know come into being a risk.

As well as all this, we keep talking about R being above and below 1, but just getting it below 1 is almost not enough in this outbreak. We need to concentrate on getting it as low as possible. I do not think that there is a threshold that we would ever want to put on it; the thing is just to minimise as many of these interactions as you can to minimise the risk.

Q869 **Katherine Fletcher:** And then we balance it with the economics.

What is coming through in your three submissions is the very broad categories that you have people in, in trying to model interactions. There is the concept of home, school and workplace, then everything else goes into the "other" bucket—leisure, travel and retail and all sorts in between. Is there any argument to say that, early doors, we could have been more sophisticated, and that that would have given us some help towards understanding the care homes, for example? Could we have added a category of people living together in groups of more than six in between home, school, workplace and other? Professor Ferguson, is it possible to do? Could we have done it?

Professor Ferguson: We are doing some of those things.

Q870 **Katherine Fletcher:** Now?

Professor Ferguson: Now, yes. One thing that we are looking at is different types of workplaces, which potentially pose different risks. The challenge is that we do not have the epidemiological data to rigorously estimate what those risks are, so we are working with economists right now, at my centre, in the Treasury and elsewhere, to build those more nuanced models, to understand the economic gains and costs of relaxing interventions on a sector-by-sector basis.

Q871 **Katherine Fletcher:** I am sorry, Professor Ferguson, but this is about people living together, not about workplaces.

Professor Ferguson: Yes, we have done similar things around those issues—we collectively, and SPI-M—around cocooning, the risks of allowing households to join together, and a number of different issues. That modelling is informative of basic principles but is very speculative. An enormous amount of uncertainty is associated with it, which is why, with regard to critically important decisions, we tend to focus on models that are somewhat simpler and where you can justify the parameters more. They have less in the way of granularity.

Katherine Fletcher: Gentlemen, thank you very much for doing this over the weekend—it is a lot of work.

Q872 **Graham Stringer:** Professor Keeling, we have heard that there was no information about care homes—it was very limited—but was it not known very early on in this pandemic, from evidence from Washington State, that once the virus got into care homes it was going to be devastating, with very high death rates? I am slightly surprised that there was not more effort to find out base information and feed it into policy. Would you care to comment on that?

Professor Keeling: It is one of these things where hindsight is a great thing. I know there were studies out there at the time. One of the biggest problems was that we were all focused on the fact that we had an outbreak that was doubling every three to four days, as we have heard. We were very concerned about losing control within the NHS, and about ICU and ITU units becoming full, and there are only so many of us and

there is only so much time. We were all focused on one area. It was mentioned—we thought about it, and we said, "Care homes are important," and we thought they were being shielded, and we probably thought that was enough.

Maybe we should have been jumping up and down and saying, "Has anyone checked care homes this week? Can anyone tell us what's happening?" But there was a lot of focus at an incredibly busy time—as Neil said, we were all working way into the night, just to get the latest set of forecasts—just to understand what was happening in hospitals. The experience from Italy, where we saw hospitals getting overwhelmed, really put the focus in that direction. With hindsight, yes, there were studies out there, but I do not think they were as prominent as what was happening at hospital level.

Q873 **Graham Stringer:** Professor Ferguson, when the models suggested that there should be an imminent lockdown, how long did it take from the models giving that clear indication that a decision should be taken straightaway to the actual decision being taken to have a lockdown?

Professor Ferguson: Lockdown-type measures were first modelled by ourselves, the London School and I think the University of Warwick all the way back in February. What clarified the need for lockdown was not the models per se but the realisation that this was an epidemic which, as we thought at the time, was doubling every four days, and greater clarity on severity: that somewhere between 0.5% and a bit over 1% was the uncertainty range of people infected who would die.

It was the turning up of those latter numbers, with the scale of impact, and the fact that the NHS would not be able to cope, that really instigated that decision.

We presented, and the London School presented, what we have now seen published in reports and papers, a little bit over nine days before the lockdown was actually implemented. That was only one input into the decision-making process at the time.

Q874 **Graham Stringer:** I accept that it is only one part of it. Given the severity and the fact that the disease was spreading more quickly than expected from the Chinese information, were you disappointed that it took nine days to take that decision?

Professor Ferguson: I said earlier that, in retrospect, I would much have preferred it to be taken a week earlier, given that many lives would have been saved.

Q875 **Graham Stringer:** In terms of the balancing of the policies that have been followed—the initial policies and then the lockdown policies—how much are they predicated on the likelihood or not of an effective vaccine being developed?

Professor Ferguson: Is that for me?

Chair: Nominate someone, Graham.

Professor Ferguson: I will have a go. They are predicated on long-term control. There is no point in having undergone the social and economic cost if you then just go back to normal and get a huge resurgence of transmission. By going down that route, something that we made very clear early on is that we were committing ourselves to a very long-term strategy. We had always hoped that we would be able to replace lockdown with more targeted measures, after getting past the first wave of transmission. But until we have a vaccine, yes, we will permanently be putting out fires with this virus and stopping large epidemics from happening.

Q876 **Graham Stringer:** My last question could be to all three or any one of you. It has been said by a number of academics that probably the best policy for the United Kingdom was halfway between what we did and the Swedish model. Is there any credibility to that view?

Professor Keeling: That is a bit of an awkward question. You are asking us to balance multiple aspects here. We are all coming here as epidemiological modellers with experience in public health, and what you are asking for is a balance between public health measures, the economy and social welfare. I do not think it has ever been made clear by anybody how we actually balance those multiple elements together. It is fairly obvious what you want to do if you want to minimise loss of life, but if you also want to balance the economy in that aspect it is very difficult, and it needs a frank and open discussion about how those two things are traded off against each other.

Professor Ferguson: I completely agree. It is about the definition of "best". I point out that the measures that we modelled in our report 9 were short of what we actually implemented. What was actually implemented was a good deal more draconian. We assumed that most people would still be going to work, for instance, with some social distancing. Matt is exactly right: the more intensive the measures you put in place in that circumstance, the lower the death toll, but the higher the economic costs. It is not our job to decide what is best.

Q877 **Chair:** I have a question on that point, Professor Ferguson. We have heard from Professor Whitty and others that deaths come from different sources. Some are the direct deaths from Covid. Some are people who cannot go to hospital to be treated for other conditions. Some, perhaps in the long term, will be as a result of the wider effects of a lockdown—some economic, some social. All three are around lives and deaths. How are those three different elements considered in SPI-M or in SAGE?

Professor Ferguson: SPI-M has now started to look at the whole issue of all-cause mortality and excess deaths. However, its focus has always been on *[Inaudible]* and controlling the transmission of Covid specifically, so that has not been a primary goal of SPI-M.

SAGE has considered the issue again in a little more detail, but only now are we really getting the data coming through to allow us to quantify those different contributions to deaths. I think that it will be several months before we see the potential negative consequences of restrictions on access to healthcare. Excess cancer deaths will happen later. They have not happened yet.

Q878 **Carol Monaghan:** I have a few questions about some of the things that we have heard this afternoon and about some of the stuff in your submissions to the Committee.

My first question is for Professor Keeling. You did a study on lockdown exit strategies. It makes the assumption that people's behaviour will return to the pre-pandemic norm. What are your assumptions for that claim? Is there an opportunity to adjust the model according to what actually happens?

Professor Keeling: I will answer the latter part of the question first. We can always adjust the model as we start to get more information on how people behave. My hope is that we will not return to the pure pre-lockdown interactions if there is a lot of infection around. However, I have heard people state that as soon as we release lockdown people will almost try to make up for lost time by going to visit everybody they have not seen. We tried to get a halfway house by making that assumption.

You have only one paper out of the suite of things that we have done. A lot of the things that we have already sent to SPI-M look at even more options than are being released in papers. We have considered a lot of uncertainty to this. What is quite clear is that we are now in a situation where R is below 1, but not vastly below 1, so it would not take much of a return to normal for R to start increasing and for there to be a second wave.

It is one of the things we ought to look at moving forward, but we need to see how people behave. This was already a question. Can we predict people? Actually, that is very difficult. We are complicated beasts, and nobody really knows how we are going to respond to any particular change. I do not think that it is feasible to do it as a predictive thing. We can only look at various scenarios.

Q879 **Carol Monaghan:** I hope that one of the things that we have learned in this is proper hand hygiene. That is one habit that I hope will stick post pandemic.

Professor Keeling, may I ask you about something you mentioned? When we were talking about the case fatality rate, you reckoned that we have it about right. We heard from the previous panel that to get an idea of the case fatality rate we need to look at the number of deaths. It looks as though we are working backwards from the number of deaths, so how on earth can we know whether we have got that figure right?

Professor Keeling: It was Nick who said that we had got the figure about right.

Carol Monaghan: Apologies.

Professor Keeling: I can try to answer the question.

Q880 **Carol Monaghan:** Maybe I should ask Dr Davies the same question.

Professor Keeling: I am happy if Nick wants to answer.

Dr Davies: We can estimate the infection fatality rate because we now have both data on the number of deaths over time and an estimate of the seroprevalence—the number of people who have developed antibodies to the virus—which gives us an estimate of the total number of infections that we have had. That gives us a much more accurate parameterisation of the infection fatality rate for the UK. That is where that comes from. It looks like it was fairly consistent with the initial estimates that came out of data from China, which I think makes sense. We were using a very large dataset from China. That is roughly how we did it.

Professor Keeling: It is worth stressing that this is not really a single number. It is an age-dependent quantity. If we could go more nuanced, it would probably depend on gender, ethnicity and underlying health comorbidities—a whole range of different options. To think of it as a single number is to simplify it down a little too much.

Q881 **Carol Monaghan:** Dr Davies, can I ask you about that? You say that it is based on data from China and other data. How do we actually know what the infection rate is if we are not testing everyone for antibodies?

Dr Davies: The infection rate is not based on data from China. It is based on data from the UK. This comes from surveys of blood donors from various regions.

Q882 **Carol Monaghan:** So these are just blind studies.

Dr Davies, you talked about lockdown early on. Your study looked at how much that would reduce contacts outside the home. You reckoned that the figure was about 90%. Has that been borne out by data?

Dr Davies: On the exact pattern by which lockdown has reduced contacts, the estimate to which you refer was always something of a guess. That particular analysis was finished in the course of less than a day. We just had to put in something that we thought could be achievable. The overall impact on contact rates, based on the telephone surveys that I mentioned earlier, seems to be about 73%. That is roughly the decrease in the number of contacts from pre-lockdown to post-lockdown. When you work that out over home versus other contacts, it is roughly consistent with the overall reduction in contacts. The difference is that people ended up reducing their home contacts as well. Of course, school contacts have decreased by more than 90%, because there are only a few kids at school.

Chair: This is your final question, Carol.

Q883 **Carol Monaghan:** It is for Professor Ferguson. Your models assumed different age stratification and case fatality rates. What were the reasons for that?

Professor Ferguson: Is the question about why different models assumed different things or whether models—

Q884 **Carol Monaghan:** Your model assumed different age-stratified case fatality rates.

Professor Ferguson: Yes. We did that because we already knew from China that this was a disease that principally affected the elderly in terms of the higher risk of death. It was important to capture that. We did the best that we could with the data available. As Nick said, we now have much better data from the UK. The headline estimates that we were using were about right. We have much more nuance on exactly which groups are at risk and exactly how it varies by age, but the numbers that we were using back in March and the estimates we came up with in March would not change very much if we used the much better estimates that we have now.

Chair: Aaron, I cut you off earlier. Do you have any further questions?

Q885 **Aaron Bell:** Thank you, Chair. I will try to be brief. I want to talk a little about immunity. All your models have made assumptions about immunity. They have to be assumptions because we do not fully understand the disease yet. Most of them work on the assumption of roughly one to two years of immunity, or maybe six months to two years of immunity. If it turns out that infections confer less immunity than that, what will the implications for the conclusions and recommendations that you have made be? I will start with Dr Davies.

Dr Davies: I am not quite sure where to begin with this one. Perhaps Professor Ferguson would be able to answer.

Professor Ferguson: This is something we have looked at. It reinforces the decision to go for lockdown. Compare that with what we called mitigation—which other people have called a herd immunity strategy—where you just get through the epidemic and try to shield the elderly. You may even succeed in that. However, if you go for that strategy, it intrinsically relies on people being immune at the end. If they are not, you are in an even worse position.

The lockdown strategy was never contingent on the population getting immunity. We think that at the moment about 8% of the population of England are immune. The figure is less in Scotland and in Wales. I am sorry; I should have said that they have been infected. They were not immune. That 8% does not make very much difference to the transmission rate. That is why we have to maintain controls. It is the downside of the strategy. However, the assumptions about immunity

mean that the modelling is quite robust. We are controlling transmission by stopping people contacting one another.

Q886 **Aaron Bell:** Professor Keeling, did you want to add to that?

Professor Keeling: I was just going to echo what Neil has said. I think that it will make a difference only when you are looking six or nine months down the line and thinking about what the eventual release of the lockdown is going to be. If we had only six months' immunity, that would just strengthen the need for a vaccine as probably the only way fully to release lockdown and to go back to normal.

Q887 **Aaron Bell:** Professor Ferguson, let us look at the other end of the scale: susceptibility. My understanding is that nearly all models assume that the whole population is susceptible. If that is not the case—there have been some suggestions that being exposed to previous coronaviruses may give you some limited protection—what will it mean for your models and the recommendations that you make?

Professor Ferguson: We always allowed for some variation in susceptibility, particularly around children. All the models represent a lot of variation in exposure in the population.

The hypothesis around cross-immunity is an interesting one. It is argued that it is mediated by T cells. The immunologists I have talked to—I am not an immunologist—tell me that that is more likely to be manifested not as a reduction in your risk of being infected overall but as a reduction in your risk of severe disease. It may contribute to our understanding of why some people get almost no symptoms and some people get very severe disease. It does not affect their chance of getting infected at all. They will still get infected, but they will not manifest symptoms and may not transmit onwards. We always allowed for the fact that "people with lesser or no symptoms" would not transmit as much.

Q888 **Aaron Bell:** So it is baked into the model anyway.

Professor Ferguson: Effectively, yes.

Q889 **Aaron Bell:** There were some criticisms of your model that it did not allow for the susceptible sub-population being depleted over time. Is that correct, or is that a misunderstanding of your model?

Professor Ferguson: It is not correct at all.

Aaron Bell: That is what I thought.

Q890 **Chair:** Would you explain why? Is it because it is included in the model or because it is not relevant to the model?

Professor Ferguson: No, it is included in the model. We always modelled the susceptible being depleted.

Professor Keeling: It is included in almost every model I could ever think of.

Q891 **Aaron Bell:** That is what I thought. I had just read the criticisms and wanted to get it on the record.

Finally, I go back to the fact that your model was initially based on influenza. You obviously had to make some changes to incorporate the fact that Covid can be transmitted pre-symptomatically—and, possibly, asymptomatically.

Professor Ferguson: Actually, that was already in the model. That happens sometimes with influenza. I should say that you do not really have a flu model—you have models for directly transmitted diseases. The last time that model was used was to model the Ebola epidemic in the DRC. It was taking out Ebola-related transmission components that took the time, not anything to do with flu. Like the other groups, we based all the parameterisation that we put into the model on Covid data, to the extent that we could.

Q892 **Aaron Bell:** That is what I was going to ask: when was it incorporated? It sounds like it already was and that it was just a question of modifying the parameters.

Professor Ferguson: Yes.

Aaron Bell: Thank you very much for your time, gentlemen.

Q893 **Zarah Sultana:** The London School of Hygiene model on the impact of non-pharmaceutical interventions and the Warwick model on potential lockdown exit strategies do not account for "individual-level variation in transmission"—that is to say, super-spreading events, as they are called. How significant is this limitation? I put the question first to Professor Keeling and then to Dr Davies.

Professor Keeling: In the context of what we were doing in the paper, it is not relevant. We were taking an aggregate view of the whole of the UK. While super-spreading events are very important when we are thinking about trying to control very small, localised outbreaks, when you scale that up to the entirety of the UK population, or even the regions that we were modelling, those events just get smeared out. Really, you can take just an aggregate view. We flagged it up in the paper as a next step, as we start to think about small-scale stochastic populations and very low levels of infection. However, for the context we are looking at, where we have a widespread UK-based infection going on, I do not think that it is that relevant. It will not change the lockdown policies greatly.

Dr Davies: I do not have much to add to that. At the London School of Hygiene, we have a variety of models that take into account different phenomena, depending on the question that is being asked. Our models looking at the effect of contact tracing, for instance, take super-spreading individual variation in transmission into account because it is important to do so in that context.

When we are talking about large-scale behavioural interventions, the aggregate behaviour is a pretty good approximation of what is going on, even in the presence of super-spreading.

Q894 **Zarah Sultana:** This question is open to all three witnesses. In your view, have the limitations of the models used in the pandemic been adequately communicated to the public?

Professor Ferguson: I think that they have been adequately communicated to policy makers. Uncertainties could probably be communicated better. It is always a challenge in terms of time. We are still working flat out. We do what we can in terms of public communication, particularly through this sort of opportunity. More certainly could be done.

Dr Davies: What I would say to the public is that models are not perfect predictions of reality. They are tools that we use to evaluate how changes in inputs can impact on outputs. I do not know whether that has been adequately communicated to the public, but all scientists are very keen that public understanding be promoted at all times.

Q895 **Chair:** Professor Keeling, do you have anything to add, or do you agree with your colleagues?

Professor Keeling: All these models are forecasts. We are used to weather forecasts. We expect them to be right today and maybe tomorrow, but the longer you go on, the more uncertainty there will be. Maybe we could use more examples like that to try to communicate with the public. As Nick said, we are all really interested in public communication of science. When you are working 24-hour days to try to get models up and running and you have teams running in parallel, you just do not have time to do public communication as well. Maybe this is a lesson for the future that we need to do more public communication beforehand.

Q896 **Zarah Sultana:** I have a final question for all three panellists. In your view, are there areas in which more modelling is needed? If so, when will that be most appropriate in order to manage the pandemic and the exit strategy?

Professor Keeling: The big thing going forward will be the test and trace strategy—trying to understand just how well contact tracing is working. I believe that all of us have models that will do that. The difficulty will be parameterising those. We have yet to see that much data coming out of the test and trace scheme. What we really need is very detailed individual level data. We have heard about the granularity of these things. The more detail we can have on how well the test and trace scheme is working, the better we will be able to do that. That will be the major challenge looking forward, for the next month or so.

Dr Davies: The work has not stopped. We are still getting requests from SPI-M and SAGE to continue to support policy making with modelling

evidence. Testing and tracing is one area we are focusing on. In the longer term, looking at vaccination strategies will be an interesting problem that we can approach with modelling. That is rather further into the future.

Professor Ferguson: I would echo Matt. I would also echo Johan Giesecke's last comment that data on understanding exactly where transmission is more likely to occur, which the test and trace scheme will give us, will be invaluable going forward in refining models and, frankly, refining policy.

Q897 **Chair:** I will wrap up the session with a reference to our first panel, which you might have heard. One of your fellow epidemiologists was concerned that there were perhaps more perspectives that ought to be brought to bear or reflected in SAGE. Professor Ferguson, you are a member of SAGE. I do not know whether you heard what he had to say. Do you think that he has a point? Is now the time, post the pressing of the emergency stop button, when a greater range of advice might be needed?

Professor Ferguson: SAGE has evolved over time quite a lot, anyhow. It is a lot more diverse now. I need to correct you. I have not sat on SAGE for the last few weeks, as people know, but it is a lot more diverse. Sir Patrick and Chris Whitty get advice from a wide range of other sources as well, so some of these criticisms have been addressed already. I still think that modelling has an important role to play, but clearly it is just one of many scientific inputs.

Q898 **Chair:** It was Professor Keeling who mentioned that we should aim to have the R rate as low as possible. Is that right? Clearly, there is a difference between its being above one and less than one. If it is less than one, it is shrinking. However, in order for it to be as low as possible, one of the things that we could do would be to lock down permanently. That would keep it as low as possible. Is that right? Is not the appropriate policy objective to keep the rate below one? There are reasons why it may not be an objective to have it literally as low as possible.

Professor Keeling: What I was trying to get at is that the value of one, or just below one, is not sufficient. We ought not to treat it as an absolute threshold or to think, "If we are below one, we are okay."

The point that I was trying to make was that, ideally, we would like to have it as low as possible, so that we get the fastest decline in cases that we can. Really, we will get out of this only by having a very low number of cases. The sooner we can get to a low number of cases, the sooner we can start to lift some of the lockdown measures.

It is a question of whether you want a very hard lockdown that is relatively short or a longer lockdown that is not quite as harsh. There is a trade-off there. Just focusing on whether R is above or below one glosses over a lot of the details. If you could get R down to 0.5, that would be

wonderful, but having it at 0.99 basically says that cases will carry on almost as they have been into the foreseeable future. One is the threshold between increasing and decreasing, but there is a lot more information that is stored in it.

Q899 **Chair:** Who should make those trade-offs?

Professor Keeling: Certainly not modellers, and certainly not epidemiologists. That is a political decision. I heard the economists being interviewed the other day. It really needs an integration of public health, probably clinicians as well, and epidemiologists. Let us bring in the economists and work out what the implications of various different strategies would be.

Q900 **Chair:** Within SAGE?

Professor Keeling: It also needs an objective function: what do we really want to minimise going forward?

Q901 **Chair:** Absolutely. Should that unified perspective be taken by SAGE, or is that for Ministers, drawing on a wide range of advice?

Professor Keeling: That must be for Ministers. It cannot be anything that can come straight from SAGE because it is not a scientific question—it is a value judgment.

Professor Ferguson: We are looking at exactly those models. We are working with economists now and feeding into those things. I completely agree with Matt. It is a policy decision.

Q902 **Chair:** This Committee has found—not least through evidence that you have given, Professor Ferguson—that the Government have followed scientific advice. Is it possible for the Government to continue to say that they have rigorously followed scientific advice if we are now saying that there are other factors besides scientific advice that need to be weighed in the balance to inform policy decisions and even the scientists agree that the Government should be taking other advice and being influenced by it?

Professor Ferguson: Yes. You can follow scientific advice, in the sense that you make policy cognisant of what the scientific evidence says and, therefore, cognisant of our best guess or best estimate of what the implications of policy decisions are. I believe that the Government have done that. However, that still gives room for the Government to choose from a menu of options. We have always put a menu of options in front of Government as policy. That is clear from the SAGE documentation and SPI-M papers that you have seen. We are not determining what policy is adopted.

Q903 **Chair:** We are very fortunate in this country in having some of the best scientists and the best academic and research institutions in the world. The Government have determinedly taken and, as far as we can see, followed scientific advice, yet the number of deaths in this country has

been higher than that in many other countries that, in some cases, are without access to the strength of our science base. Professor Ferguson, in your view, is it too early to assess how well we have done?

Professor Ferguson: I think that we can justifiably start that process now. We are at the tail end of the first wave. It is probably too early objectively to evaluate the advisory and policy processes that led to the decisions that were made being made. To some extent, the very rigorous, well-established and sophisticated policy advice structure that exists within the UK Government in crises may have led to a certain degree of caution in decision making—balancing evidence, balancing certainty and uncertainty, and being very aware of costs and the risks of second waves. Frankly, I think that policy makers in many countries were not aware of issues in that way when they made the decisions that they made. There are almost certainly lessons to learn from that.

Q904 **Chair:** Do you expect the overall verdict on our relative performance to change between now and, say, a year's time?

Professor Ferguson: Potentially, we will see issues in Latin American countries. We are already seeing the situation in Brazil. Within high-income countries, I do not think that our position will necessarily change in a European setting. First of all, you should always compare per capita deaths. In a European context, it is a little too early to say what will happen in Sweden, but in other countries I do not think that they will see a change.

There is a big open question regarding the United States and measures that are being adopted there. They are probably at a higher risk of a significant second wave than most European countries.

Q905 **Chair:** Given that there are some decisions that will continue to need to be taken, if the verdict is unlikely to change in any material respect, notwithstanding our science base and the structures that we have, are there any lessons as regards the structure of scientific advice that we should take now to inform those decisions, or do we need to reflect further on that?

Professor Ferguson: I have been too close to it to be objective enough necessarily to be the person to answer that question. I have observed how certain things have happened, but I have my own view. I think that it requires an objective, external view really to learn those lessons well.

Chair: I am very grateful to all the witnesses. We have overstayed our time a little, but the discussion was very important and informative. It is the purpose of the Committee in undertaking this inquiry to capture evidence that can be used for a long-term look back, but also, where it is possible, to learn lessons on the way that will be germane to decisions that are yet to be taken. Even if they are sometimes challenging and uncomfortable lessons, it is appropriate to be able to take them.

That is the spirit in which we have conducted this inquiry. I am very

grateful for your evidence today. I am particularly grateful to you, Professor Ferguson. It is the third time that you have appeared before the Committee. We are very grateful for that and to your colleagues at all three institutions for the work that they continue to do.