HOUSE OF COMMONS

Science and Technology Committee

Oral evidence: UK Science, Research and Technology Capability and Influence in Global Disease Outbreaks, HC 93

Tuesday 26 October 2021

Ordered by the House of Commons to be published on 26 October 2021.

Watch the meeting

Members present: Greg Clark (Chair); Dehenna Davison; Katherine Fletcher; Rebecca Long Bailey; Carol Monaghan; Graham Stringer.

Questions 2498 - 2621

Witnesses

I: Professor Andrew Curran, Chief Scientific Adviser and Director of Research, Health and Safety Executive; and Professor Mario Mondelli, Professor and Director of Infectious Diseases, University of Pavia.

II: Professor Sir Andrew Pollard, Professor of Paediatric Infection and Immunity, University of Oxford and Director, Oxford Vaccine Group; and Dr Sharon Alroy-Preis, Director of Public Health Services, Israel.

III: Professor Sir Stephen Holgate, Medical Research Council Clinical Professor of Immunopharmacology and Honorary Consultant Physician within Medicine, University of Southampton; Professor Lucy Chappell, Chief Scientific Adviser, Department of Health and Social Care; and Dr Thomas Waite, Interim Deputy Chief Medical Officer, Department for Health and Social Care.

Examination of witnesses

Witnesses: Professor Curran and Professor Mondelli.

Q2498 **Chair:** The Committee is now in session. The Science and Technology Committee is today considering next steps in the handling and response to the Covid pandemic as we look to the autumn and winter. We will be hearing from leading scientists in the UK and internationally to consider their latest perspective and advice.

First, we will consider the latest knowledge on how Covid is transmitted. Then we will turn to the role of vaccines, and, in particular, the role of boosters and vaccines for children. Finally, we will consider the plans that are proposed for this winter, and beyond, here in the UK.

I am very pleased to welcome our first witnesses, who are with us in the room. Professor Andrew Curran is the chief scientific adviser at the Health and Safety Executive. He has been an active participant in SAGE during the pandemic. I am very pleased to welcome from Italy Professor Mario Mondelli, who is professor of infectious diseases at the University of Pavia. We are very grateful for your attendance today before the Committee.

I will start with a question to both witnesses. Professor Curran, what do we know about the principal routes by which Covid is transmitted?

Professor Curran: Thank you; good morning. The three principal routes by which Covid is transmitted are air, person to person, and surface contamination. Obviously, as the pandemic has progressed, we have gained more knowledge about those different routes, but certainly from the evidence we are producing under the National Core Study on transmission we believe that all those three routes still have a part to play. Essentially, the transmission of Covid is a continuous risk, and the factors that might lead to that risk developing into an infection are very complex. Therefore, we do not want to rule out any one route because we do not think the evidence is strong enough to do that at present.

Q2499 **Chair:** What do we know about the importance of each of those three relative to each other?

Professor Curran: Again, it has been a bit of a changing picture over the course of the pandemic. At the start, there was probably an emphasis on surface contamination, but as the pandemic has progressed it has become very clear that airborne transmission of small particles is absolutely critically important. It is very difficult to say that one route is more important than another because the key issue is that we are dealing with complex systems, and, when risk factors accumulate in particular settings, the mix of risk factors could mean that a different route of transmission is more important in that particular setting compared to another setting.

By that, I mean that you will get, for example, different demographies coming to a setting depending on where the setting is located; there will

be different background rates of infection; and there will be different mitigations in place. There will be a whole range of complex factors that ultimately determine whether transmission will occur in that particular environment.

Q2500 **Chair:** We are over 18 months into the pandemic now and there have been millions of cases around the world. Surely, we must be in a position, given the volume of evidence that is available around the world, to be a bit clearer about what the most important risks of transmission are.

Professor Curran: I come back to the fact that it depends on the specific circumstances. I will give you some examples. As part of our National Core Study, we have been looking at outbreaks, because that is really the best available information you can get about what is going on in the real world. We felt that getting real-world data was critical in understanding exactly what was going on. It soon became apparent that, for example, if you looked at the outbreaks in similar settings in different locations, you saw different things. You would not see, for example, the same attack rate if you looked in warehouses in the north of England versus the south of England. It varied quite significantly. Therefore, different things are going on in those work environments.

What we believe is that depending on the complex mix of issues—and that can be the mitigations that have been put in place by the work environment, behavioural issues, demographies, et cetera—you will get these differential patterns of transmission. Therefore, we cannot say for definite that one route is more important than another in any one situation. What we can say is that we think the airborne route has become more important and is clearly, if pushed, the most critical, but I would not want to say that we could rule the others out, and, as I say, it would depend on the specific circumstances of each incident that you are looking at.

Q2501 **Chair:** Thank you. Professor Mondelli, given your research in Italy, but also looking around the world, what is your perspective on what are the principal routes of transmission, and, particularly, the relative risk of each route?

Professor Mondelli: First of all, thank you for inviting me, and hello to everybody. I essentially agree with Professor Curran. Airborne transmission is the most important way of transmission of Covid-19—SARS-CoV-2.

We have done some work on transmission through fomites—inanimate surfaces, as you probably know—and what we found was that in the real world the probability of transmission through inanimate surfaces was really not important and probably not present. The virus was present in minute amounts but those minute amounts were unable to transmit the infection in vitro to susceptible cells. I believe that transmission via fomites—inanimate surfaces—is really a minor way of transmission, but

we should not lower our guard, and we should keep on considering those ways of transmission as possible even though they are highly improbable.

Q2502 **Chair:** Thank you. Before I turn to my colleague, Graham Stringer, was that based on research that you conducted or supervised in Italy, and in what settings, or is that based on an appraisal of the international literature?

Professor Mondelli: We did work in the field, and specifically in ordinary infectious disease wards, dedicated emergency rooms and sub-intensive wards—very high-risk places. We did routine disinfection and cleaning procedures using chlorine once a day, but we did not do any cleaning of the walls regularly. The cleaning was performed essentially of the floor. There were several other objects that we looked at by swabbing with a specific floq that is currently used as a nasopharyngeal swab. We used transport media to swab all the surfaces, including computer keyboards, cell phones, consoles, infusion pumps, cupboards, and gowns worn by medical staff and nurses. We found some traces of virus on the CPAP helmet, which is what you would expect so close to the airway of the patient. I would have been surprised if we did not find any trace of viral irony.

We did two studies, and the pathology was quite similar. I want to emphasise the differences in the experimental approach in early studies by American groups. In the lab, people sprayed a high concentration of the virus on to surfaces like copper, steel, plastic and cardboard, and ended up infecting with a high titre of virus on those surfaces, which took several hours, if not days, to disappear. That was in an experimental setting and not a real-life setting, which is the one we have to do.

Q2503 **Chair:** The early experiments were putting very high levels of contamination—

Professor Mondelli: In the laboratory, yes.

Q2504 **Chair:** —in the laboratory, but the settings in which you analysed were—

Professor Mondelli: These were real life.

Q2505 **Chair:** —real life, but in hospitals where there was a significant risk of Covid, you found that it was only in the CPAP—a kind of oxygen helmet—

Professor Mondelli: A helmet, yes.

Q2506 **Chair:** It was only that that had appreciable contamination.

Professor Mondelli: Contamination. Despite swabbing and seeding into susceptible cells, we were not able to grow any virus from those isolates. Obviously, we did not do any investigation in malls, shops or department stores. That would be another interesting setting. I believe that the evidence that we produced shows that the common disinfection procedures are enough to avoid transmission through fomites.

Chair: Thank you very much indeed. That is very clear and helpful.

Q2507 **Graham Stringer:** Professor Curran, my apologies, I will have to leave soon after these questions. Can you tell us what PROTECT has found to be the highest risk workplaces?

Professor Curran: It is a very interesting question. The information has changed over the course of the pandemic. It depends how you quantify that risk. The early studies looked at epidemiological evidence and excess mortality in certain occupations using census data from 2011. That demonstrated that there were a number of occupations that had an increased risk, but those calculations did not correct for other potential risk factors such as socioeconomic status, ethnicity or other various issues. Again, as part of the National Core Study, we have done some analysis where we have looked at some of those other risk factors, and we demonstrated that the association with work fell quite significantly once those risk factors were taken into account. While it did not disappear completely, it dropped by a significant amount such that the excess risk was not doubled, which is one of the approaches that is used to determine an excess of risk.

The early work that was done gave us an indication of where we needed to put more attention and collect more data. The more recent work that has been done has demonstrated that, while there is some additional risk, for example, in transport workers, meat processing workers or people who are exposed to members of the public, it is not as great a risk as it was originally thought now that we have done those more detailed analyses.

It also comes back to the point that I was making earlier. It is very difficult to say that one workplace is riskier than another. A complex mix of factors actually determines that risk, and understanding how those risk factors coalesce in particular environments is becoming increasingly important. To give you an example, we know that there are areas of enduring prevalence of Covid in an arc from the north-west through Yorkshire and the Humber down through the midlands. Explaining why that is the case and has continued to be the case is something we are looking at, because that will help us to unpick how all of these different risk factors are coming together to cause, in particular circumstances, transmission to be greater and the cases of Covid to be greater.

It is quite a complicated picture. I would not want, therefore, to say that this workplace is more dangerous than that workplace because that would not be an accurate reflection of what is really going on. Clearly, some occupations have an increased risk compared to others from the epidemiological work that has been done.

Q2508 **Graham Stringer:** Let me ask you specifically then. About six months into the epidemic, I wrote to the ONS and asked them about the death rates for people working in supermarkets—shop workers, effectively. They wrote back and said that, for men, the rates of death were 75% higher than you would get in their cohort, and for women it was 65%. Are you saying I cannot draw any conclusions from that about the safety

of food workers in supermarkets?

Professor Curran: Obviously, it depends on the analysis that the ONS did and how they corrected for confounding factors. I am not aware of what was done there.

Q2509 **Graham Stringer:** I think they did correct for socioeconomic background.

Professor Curran: Okay. If they corrected for socioeconomic background and ethnicity, clearly, that would indicate that there was an increased risk in those groups. From any of the other work that I have seen, I am not sure that supermarket workers come out particularly at risk, but, clearly, the thing that would be of concern for supermarket workers, potentially, is that the exposure to the public, who may not be taking precautionary measures such as wearing face coverings, et cetera, might provide the opportunity for them to be exposed to more virus. It depends on what is going on in the supermarket itself, how the controls are being managed, what the ventilation looks like, and what specific mitigations the supermarkets have put in place, which vary quite significantly.

Q2510 **Graham Stringer:** I can understand your caution in trying to put figures on the different routes of transmission, but would it be safe to say that surface transmission is close to zero?

Professor Curran: It would not, no. I was very interested in what Professor Mondelli talked about. I agree totally that measuring in the real world is really important, and that is something that we have been doing when we have been going out and investigating outbreaks. To give you an example of a recent outbreak that we investigated in an office, we went in and took samples from all around the office, and we found very high contamination of SARS-CoV-2 mRNA in the security bit. The security was outsourced to a particular company, and they were using a different risk assessment model. We found a lot of evidence of viral contamination in the office that they were using. We found it on, for example, the handles of the windows in that office as well.

Therefore, that is why I would not say that there is zero risk from surface transmission, because we found mRNA—which is not the same as live virus; it is an indicator that there is a lot of virus in the space—on a lot of surfaces in that particular example. In many other examples we have been to, such as food processing factories, you can find it not so much in the work area but in the locker rooms, the canteens, the toilets, et cetera—the work-associated areas. That is why I do not think you can say with certainty that surface transmission can be ruled out.

Q2511 **Graham Stringer:** You have evidence of contamination but not transmission. Is that fair to say?

Professor Curran: And it would be very difficult to do that. To demonstrate that it was purely surface rather than air would be a very difficult thing to do.

Q2512 **Graham Stringer:** Right. Given that air transmission appears to be the main route, do you have a view on the effectiveness of masks?

Professor Curran: It depends what you mean by masks. I would divide them into two categories. First of all, I will talk about face coverings, which are the fluid resistant surgical masks that you see primarily out and about. They are good for source control. If I am wearing one of those, I am protecting other people from me, essentially. It probably gives me a little bit of protection from other people, but primarily it is acting as source control. If you then move into what we, in HSE, would describe as respiratory protective equipment, which has proper standards and what have you, the different standards will determine the level of protection that you get. Typically, in a high-risk area in a hospital, you might be using an FFP3 respiratory mask and wearing that after having been face fit tested. You cannot just put one on and assume it is going to work; you have to test to make sure that it fits your particular face and provides you with the protection that you need.

So, yes, masks have a role to play, but it is really important to understand what they do and what they do not do when working out how they can help you to control the risks that you are trying to manage.

Q2513 **Graham Stringer:** Can I put my final question to Professor Mondelli? Possibly this winter, Professor Mondelli, we are facing not only infection from Covid but from influenza. Can you briefly tell us the difference between the transmission routes of influenza and Covid? Will the non-pharmaceutical interventions—masks and other precautions—be the same for flu as they are for Covid?

Professor Mondelli: Thank you for asking. Essentially, flu is transmitted very similarly to SARS-CoV-2, with probably more risk played by inanimate surfaces. There is no real evidence supporting that; it is just an impression and data coming from early studies.

Barrier methods such as masks play a significant role. In Italy, for example, we saw a real drop in transmission of flu during the first lockdown. Social distancing and masks were enough to really demonstrate a drop in other respiratory viruses particularly. I believe that flu shots are also very helpful. The response rate in terms of vaccination of the entire population is very minimal compared to the SARS-CoV-2 immunisation. In Italy, for example, for reasons that are not completely apparent, before Covid we had only a 33% response rate for flu vaccination, even in the elderly. It is probably a cultural attitude. People thought that flu was not so dangerous, but everybody knows that flu can be dangerous in vulnerable people. Several flu waves have been witnessed, or at least four different waves, due to antigenic drifting demonstrated since the first flu pandemic.

Coming back to the barrier method, I believe that masks are very important, and there is evidence that masks protect from respiratory viral infections. A large study conducted in India on Covid showed that even surgical masks are not really perfect. Professor Curran said they are enough to significantly reduce infection. Obviously, FFP2 masks are much better, but they are heavy to wear all day. We do it in the wards, of course, and when seeing outpatients because we have to, but going beyond six hours was asking too much. This is not as difficult for surgical masks, which are protective, as I said. I believe this is the answer to your question.

Graham Stringer: Thank you.

Q2514 **Chair:** Thank you, Graham.

Professor Curran, you mentioned research that you have done over the past year. Does the Health and Safety Executive consider formally international research in making its recommendations?

Professor Curran: Absolutely. For example, one of the things that we did very early in the pandemic was activate our international networks, one of which is the Partnership for European Research in Occupational Safety and Health. We had an agreement with all of the countries involved in that—there are 13 countries and 14 different institutions across those 13 countries—that we would share information on a very rapid basis. In fact, our colleagues in Austria set up a platform to enable us to do that. That meant that we could get very rapid access to information that was emerging, for example, on masks. There were some questions early doors about reuse of masks and cleaning of masks. We could see what colleagues in Switzerland were doing because they had started that work earlier. That was extremely helpful to us. That work and collaboration has continued.

Similarly, I chair something called the Sheffield Group, which is an international network of national health and safety labs. We asked questions of that global network around various issues during the course of the pandemic and got an international perspective on what we were doing. That international view has always fed into what we do in a really practical, focused way.

Q2515 **Chair:** Is the work that Professor Mondelli described something that you have made an evaluation of?

Professor Curran: Certainly, the work that Professor Mondelli has talked about, I think, has been considered recently by UKHSA, which has just published a systematic review of face coverings, which included international members on its steering panel.

Q2516 **Chair:** I am thinking particularly of surface transmission.

Professor Curran: Certainly, in our National Core Study, we are reviewing the literature all over the world at all times. That plays into not just the interpretation that we make but also directs some of the

experimentation that we do to make sure that we are not just focusing on a very small view of the world but taking in that global perspective. Absolutely.

Q2517 **Chair:** Professor Mondelli, you described your study that found that there was very limited potential for transmission in a hospital setting from surfaces from fomites. Professor Curran mentioned that there was the presence of mRNA detected—not necessarily transmission—on surfaces. Do you have an explanation for why that might be the case?

Professor Mondelli: An explanation for why mRNA was present on surfaces?

Q2518 **Chair:** The implications of that. Would the presence of mRNA lead to concerns about transmission? How does that compare with your study in the hospital?

Professor Mondelli: We did not look for mRNA in our initial studies last year, but, certainly, the presence of mRNA would suggest that the message for synthesising proteins is there. Whether this mRNA is viable in terms of actually producing viable viruses has not been shown. The only experiment or test that we have available to suggest that the RNA that is found on surfaces is actually able to promote the spread of the virus, at least in vitro, is the infection of susceptible cells. These are vero cells—a special clone of the vero cells from a monkey kidney—and these cells can be used for a variety of reasons, including calculating the neutralisation potential of antibodies elicited by the vaccine that we have available against Covid.

RNA—ribonucleic acid—is susceptible to degradation compared to DNA, which is much more resistant. RNA is probably very quickly denatured and unable to transmit the infection and transduce the proteins that are essential to form the complete virus and to give rise to a proper replication procedure in the construction of new variants. It is interesting that mRNA has been found on surfaces, but this, unfortunately, does not prove that such mRNA may be responsible for transmission for all the reasons that I have just mentioned.

Chair: Thank you.

Q2519 **Carol Monaghan:** Professor Mondelli, can I ask you a little more about that? You talked about the mRNA degrading. How quickly would that happen?

Professor Mondelli: From minutes to hours. It depends on the environmental conditions. For example, humidity and temperature play a significant role. UV radiation may be another factor. We keep on saying that summer will reduce transmission of SARS-CoV-2. Unfortunately, this applies to particular UV rays—type C UV rays, and not the common type A and B that reach the ground on earth. Type C rays are largely blocked by the ozone barrier.

I said that RNA is much more prone to degradation. In fact, other DNA viruses can resist the environment more, such as hepatitis B, which could be sent in very standard packaging around the nation. Several years ago in Italy, there was not much control of transmission. Fifty years ago, there were no specific containers, and we sent blood samples without any— *[Inaudible]*—and it could elicit an infectious virus. This is usually not possible for an RNA virus. It varies a lot even among RNA viruses.

Q2520 **Carol Monaghan:** Thank you. Professor Curran, given what we have heard from Professor Mondelli this morning about his studies and other information, are we focusing, or have we focused, too much on surface transmission?

Professor Curran: We could have focused more on airborne transmission at the start, definitely. We have corrected that. There has been a big move towards identifying the need to improve ventilation, for example, to make sure that spaces are appropriately managed from an occupancy perspective. I do not want to overemphasise the potential for surface transmission, but I also would not want to rule it out. As Professor Mondelli says, identifying mRNA on a surface is not evidence of transmission, but we have also been able to grow live virus from surfaces that have been swabbed outside the hospital environment. This work has been done by one of the groups within our National Core Study consortium using samples from homes, for example, and remote controls, telephones and that kind of thing. They have been able to grow live virus.

Therefore, that, if you like, is the missing link between whether a live virus can be picked up from surfaces. Yes, it can in those studies, as yet unpublished. That is why we have not majored on that yet but that is something that will come out over time. It is very difficult to measure live virus on surfaces. It is also very difficult to measure it in air samples. That step between measuring a marker of exposure, the messenger RNA, and demonstrating that that is associated with live virus is a very difficult thing to do, not just for surfaces but also for air, and only a small number of groups around the world have been able to do that.

Q2521 **Carol Monaghan:** You said a few times, and Professor Mondelli said it as well, that evidence of virus does not mean evidence of transmission, which is important. The WHO has said that there are no specific reports that have directly demonstrated fomite transmission. Does that mean that there have been zero cases of Covid picked up from a surface?

Professor Curran: The challenge is finding an example where you could isolate the fomite route alone, because to do the kind of work that would enable you to demonstrate that is, as I said earlier, very complicated. If you work on the basis that transmission is a continuous risk, working out where you got it if you suddenly test positive is very difficult because it could be in any one of any locations that you have been subjected to from the minute you get up to the minute you go to bed. Being able to demonstrate very specific issues like that depends on your ability to identify some very specific exposure scenarios where, for example, every

other route had been eliminated and you could say, "Yes, definitely, that is fomite transmission."

I am trying desperately to think of one, but I cannot off the top of my head, and that is the problem. It is very hard to be able to isolate very clearly, given that transmission is a continuous risk and those three routes of exposure are all absolutely possible.

Carol Monaghan: Thank you.

Professor Mondelli: May I add something?

Carol Monaghan: Yes, please.

Professor Mondelli: There are two critical factors here as well, and I agree with Professor Curran. One is timing. Are we sure that the virus that can be isolated from surfaces has been there for some time or has just been spit by somebody who sneezed on the surface and it is quite fresh? It is difficult to determine for how long the RNA has been there. It has probably been there for only a few minutes. My feeling is that the virus, if transmitted at all by surfaces or fomites, should be freshly spit on the given surface to be infectious. That is one point.

The other point is that, to demonstrate that a particular clone of virus has been responsible for transmission to a third person, you have to sequence the virus retrieved from the fomite or the surface and the virus that is present at any given time in a patient who has allegedly been exposed to the surface. So, as you can see, it is extremely difficult.

Q2522 **Carol Monaghan:** That patient might have touched many other surfaces as well, so I can see that that would become almost impossible.

Professor Mondelli: Yes. You can model everything, but when you come to hard evidence it is really very difficult to demonstrate that transmission has occurred from surface to a patient.

Q2523 **Carol Monaghan:** Professor Curran, one of the pieces of evidence we took early on was about the composition of SAGE. We found that there was overemphasis of certain types of experts and probably less of others—for example, engineers. We know that engineers have been instrumental in demonstrating airborne transmission. Did we consult them early enough in the pandemic?

Professor Curran: It is not really my role to comment on SAGE. What I can comment on is the fact that I think it was in about April last year that I was asked by Sir Patrick Vallance to set up a subgroup of SAGE called the environmental and modelling group, which I did with Professor Cath Noakes, who has already appeared at this Committee last year with me.

We established a team of experts, including building engineers, mathematical modellers with experience of computational fluid dynamics who could look at the spread of virus, and microbiologists with experience

of how to control transmission through, for example, air-cleaning devices. We pulled together a world-class team that also had access to international collaborations and networks that they had already to answer some of the difficult questions about engineering controls and what we understood about the movement of aerosols in rooms—the physics behind all of that. That information was fed into SAGE and has been regularly since April last year. SAGE has had access to a very significant group of individuals with extensive experience in this space and has made use of that information. If you look at the number of SAGE papers that have been produced since then in that space, it did it.

Q2524 **Carol Monaghan:** You are saying April last year, but it took until April this year for the WHO to make a statement about aerosol transmission. In your view, why do you think it took it so long?

Professor Curran: I would leave it to the WHO to comment on that. If you look back, as I did over the last few days in preparation for this meeting, to what we said in the very first SAGE paper that we contributed from that environmental and modelling group, a lot of what we said then is still relevant today. We talked about the airborne route. We talked about how you mitigate using a risk assessment methodology backed up by a hierarchy of control. All of those things are still very relevant and also have more evidence to back them up.

Q2525 **Carol Monaghan:** That information was conveyed well, and people understood that, but why did the WHO not update its guidance?

Professor Curran: Obviously, I am not party to how the WHO goes through its decision-making process. We have been able to provide evidence to show that that airborne route was very important right back in April 2020.

Carol Monaghan: Thank you. Thank you, Chair.

Chair: Thank you very much, Carol.

Q2526 **Rebecca Long Bailey:** Thank you both for coming to speak to the Committee today. I will start with a very general question. I am sure you will both be aware that, as I understand it, the UK currently has one of the highest weekly rates of transmission in the world. How should the Government plan and prioritise interventions to lower transmission risk at this time? I will start with Professor Curran.

Professor Curran: The numbers are there for everybody to see. One of the things that I can speak to, as HSE's chief scientific adviser, is that HSE has provided practical advice throughout the pandemic and continues to do that to make sure that people have access to the best available guidance as to how to put in place the most effective mitigations to manage exposure via those three routes, starting with, as I have already said, the risk assessment process.

That is a very useful starting point for anybody to think about what they do, how they do it and to recognise that the risk is there continuously. There was a bit of an issue at the start of the pandemic where people were very keen to identify a particular work activity and focus on a particular element of work without thinking, "How do I get to work? Do I meet my kids at the school gate?", and all those other things that can introduce transmission risk.

That is why the guidance has to include every citizen. Every citizen needs to be making an informed decision about what they do and why they do it. From an HSE perspective, we try to make that practical and pragmatic. For example, our ventilation guidance is extremely helpful because it is a very complicated issue to work out whether your ventilation is working or not. While the cases are high, the information needs to be made available to people so that they can make informed choices.

Ultimately, I suppose it depends on what the objective is. Is it to get to a certain number of cases, is it to get to a certain number of hospitalisations, or is it to get to any other end point on that journey? Case numbers on their own are an indication of how much virus is in the environment, not how much damage is being caused by the virus per se. It is a complicated set of issues that leads you to a particular path, and that is where the decision makers come in as opposed to scientists, who are just providing the evidence.

Q2527 **Rebecca Long Bailey:** Thank you.

Professor Mondelli, what should the UK Government be doing at this time to lower transmission risk?

Professor Mondelli: I believe that reinstating the obligation to wear a mask in closed environments is very important. Try to improve the vaccine acceptance. I think you have about 78% or fewer people who have received two doses. Both should reach 90%. We are at just about 80%. We have a less dangerous situation at the moment where you hope you would manage like that.

There should be social distancing and wearing of masks inside. Avoid grouping of too many people and implement these measures. Showing evidence of vaccinations is important. We have a huge discussion about exhibiting green passes for work or having access to restaurants, theatres and cinemas. Despite all these protests, we need to be firm and implement that very simple guidance, which is common sense but absolutely essential to control the pandemic.

Q2528 **Rebecca Long Bailey:** Thank you, Professor Mondelli.

Professor Curran, you mentioned HSE risk assessments. I understand that guidance was issued by HSE when restrictions were gradually eased. Are you content that the current mitigations that are in place in workplaces, if indeed they have been followed by particular workplaces, are sufficient to limit transmission at this time?

Professor Curran: One of the things that HSE has been able to do during the course of the pandemic is respond to a changing evidence base and provide updates to guidance as appropriate, working closely with BEIS, which produces the "better working" guidance. That ability to work across traditional departmental boundaries has been very good during the pandemic. Certainly, my CSA colleagues have worked very closely together to make sure that we are getting the right evidence into the right bits of the system.

To check whether things were actually being done appropriately, HSE instigated a series of spot inspections. Therefore, we have had boots on the ground, and we have had people calling companies and following that up with visits. You can start to see from something like 330,000 spot inspections—I cannot remember the exact number—that the vast majority of organisations, well over 90%, that we have been to, which have either come to us because people have raised concerns or because they have been identified as being in a particular risk category, are doing things that meet the guidance. Our inspectors have identified that the majority of employers that they have spoken to have a desire to do things right. That is not to say that everybody gets it right, but certainly from my perspective I have been very impressed as to how people have put in place what are simple but effective things to reduce the opportunity for transmission.

We have seen it again in the work we are doing in our National Core Study where we go out to real workplaces to see what is going on. The best solutions are those that are co-created by the employers and the employees because they are the best people to work out what they do, where the risks are, thinking about the whole process from start to finish— for example, by rearranging a production line, providing a different rota system or making sure that breaks are taken differently. There are a whole range of things that people can do that make a massive difference. Where it has been done well, it has made a big difference to the potential for transmission. That is not to forget that in some workplaces it has not been done as well as anybody would have liked. The vast majority have done a great job, but some clearly have not.

Q2529 **Rebecca Long Bailey:** That is very helpful. You mentioned a case earlier where in one particular workplace a contractor had been following a different risk assessment model from the workers who were employed in another part of the office. Do you think that the HSE's guidance needs to be more stringent, and, if so, what would you like to see change going forward?

Professor Curran: It illustrates some of the complexities of modern workplaces where you probably do not always have the simple single controlling mind within an organisation. You might have a number of different contractual relationships. You might have a different caterer.

You might have a different security company. You might have a different facilities management company.

One thing that Covid has done is to shine a spotlight on how those different organisational arrangements, if they are not properly thought through, can lead to issues around what I would call the interfaces in these complex systems. It is really important that when it comes to something like Covid, which does not respect contractual arrangements or organisational differences, everybody is on the same page and everybody is thinking about the risks, which will be the same for all the people working in that space, and therefore the risk assessment process needs to take that into consideration.

That is a learning point for everybody in the context of how we manage risks going forward because the world of work has changed, and will continue to change, and we need to think how the processes that have been tried and tested since 1974 can be made appropriately to manage in that situation. My experience has been that the health and safety workout has provided a fantastic bedrock for this country to have one of the safest working environments anywhere in the world. Therefore, it has a good track record and probably just needs more conversation about how to make it work in those complex circumstances.

Q2530 **Rebecca Long Bailey:** Thanks. That is really helpful. On the issue of spot inspections, you mentioned that there had been 330,000 spot inspections so far by the HSE. Could you confirm the date when those spot inspections began, and if you are able to—you might not have this information—what were the key determinants of how a business was selected for a spot inspection? Was it limited to certain sectors that were more at risk, or was a wide spread chosen deliberately to cover all sectors?

Professor Curran: That is probably a question better asked of my regulatory colleagues. What I can say is that as of 15 October we have done 330,229 spot checks and investigated 741 outbreaks as HSE. That is a huge amount of information. That information is also now feeding into our National Core Study because we recognise that the data that has been collected as part of that process is really important.

As Professor Mondelli and I have said, that view of the real world is absolutely critical to understand what is actually going on as opposed to what we think might be going on. That is why I always come back to the point that, with transmission being a continuous risk and with workplaces being very complex environments, you really have to get into the detail to work out exactly what is going on. You cannot necessarily say that all meat processing factories are the same or all offices are the same. What you need to understand and what our study is starting to unravel now are the key risk factors that might come together in different combinations and different workplaces to cause an increased risk to those workers and employers who are in that space.

Q2531 **Rebecca Long Bailey:** Thank you, Professor Curran. I can see you have a lot of graphs there. Do any of them show what proportion of those workplaces of that 330,000 were in compliance with HSE guidance and how many were not?

Professor Curran: The way that the spot checks work is that they look to make sure that the workplace is complying with the public health guidance or the "working safely" guidance. The printing is very small, but from memory it is about 96%. That is why there is good evidence that UK workplaces have taken this seriously. I totally accept that 330,000 is not all workplaces in the UK. Of those workplaces that we have been to, both from telephone contact and actually going and looking at the workplace, the vast majority—greater than 90%—have put effective mitigations in place that comply with the guidance laid out.

Q2532 **Rebecca Long Bailey:** Thank you very much.

Finally, Professor Mondelli, as I referenced earlier, the UK has one of the highest weekly rates of reported cases in the world. Are you aware of what mitigation measures other countries have in place that are successful at the moment in lowering transmission rates?

Professor Mondelli: Most of the countries are following what we have been doing in Italy—wearing masks indoors and trying to reach all the population with vaccines. There are a number of people in Italy who are "no vax" and some people who are uncertain about vaccines. While you cannot do anything about convincing "no-vax" people to vaccinate themselves, you can do a lot with teaching and information to try to convince the uncertain people. We see quite a lot out there. We believe we have 3 million people who have not been vaccinated who could have been.

Another important point is to vaccinate children from the ages of five to 12. It is common sense that children, at least last year, had a very mild disease but, in fact, some complications for children can be avoided by vaccinating them. The adverse effects are quite manageable and not life threatening.

We have something called a green pass—I am not sure if you have it in Britain; it seems that you do not—which is evidence of vaccination or evidence of having a nasopharyngeal swab in the previous 48 hours. Using a rapid swab is probably not enough to be absolutely sure that the subject is not infectious.

The elementary guides of social distancing, wearing masks, particularly indoors, and getting as many people vaccinated as possible, are three very important issues that can be responsible for mitigating transmission of the virus.

Rebecca Long Bailey: Thank you. That is very helpful.

Q2533 **Chair:** Professor Curran, at the beginning of the pandemic, we were

seeing through a glass darkly. We did not know what the principal groups of transmission were. We are nearly two years on and we know more about routes of transmission. Has the HSE guidance on fomite transmission—surface transmission—changed?

Professor Curran: No, I do not think it has. It has not because we cannot rule it out as a route of transmission, as I have said throughout.

Q2534 **Chair:** You say you cannot rule it out, Professor Curran, but the work of HSE in many other areas of national life involves making some judgments as to what the appropriate level of risk is and applying expertise to that. Are you following that practice here, or are you following an approach that is providing ultimate caution so that we all have these hand wipes and sanitisers? You have them there. That is costly at the margin to people, to organisations, to businesses and to the environment. Are you being rigorous in maintaining the same restrictions now after nearly two years when we know so much more about surface transmission?

Professor Curran: It is important to recognise, No. 1, that it is the responsibility of the duty holder to assess the risk that their activities are generating. If, for example, the work that they do does not generate the risk of surface transmission, they do not need to put in place mitigations for surface transmission. HSE does not mandate—

Q2535 **Chair:** I understand that, but my point is—and Professor Mondelli has been very clear—that in a rigorous study there was not any evidence of significant contamination in a hospital setting. The evidence at the beginning—which is perfectly reasonable to follow—was experimental. Our colleague, Graham Stringer, pointed out that the WHO has not been able to find any evidence of fomite transmission. We discussed that it might be hard to do that. It seems to me that nearly two years on, surely, HSE should be looking at this again. Do you have plans to do that?

Professor Curran: Obviously, we keep the evidence under continuous review.

Q2536 **Chair:** Well, do you? Tell me what review you have conducted recently around that.

Professor Curran: For example, obviously one of the issues we have looked at recently—bearing in mind that there is no evidence for airborne transmission of the same kind when it comes to fomite transmission—is that you cannot measure live virus in the air very easily.

Chair: Indeed.

Professor Curran: You are making judgments there as well as to the relative importance of those two routes. Certainly, for example, we have looked very significantly at our ventilation guidance. We have looked very significantly at our guidance on carbon dioxide monitoring.

Q2537 **Chair:** I am talking particularly about surface transmission.

Professor Curran: In terms of surface transmission, we keep the evidence base under review, and we have identified—

Q2538 **Chair:** Can we see the papers that you have taken on this?

Professor Curran: All the information that we would use are the SAGE papers. The SAGE papers that look at the route of transmission, the risks of transmission, the virus characteristics, et cetera, are the bits of information that we will use to determine whether a review of the evidence that we are undertaking in terms of our guidance will be required. As things stand, we have not seen, from a surface transmission perspective, any need to change that guidance at present.

Q2539 **Chair:** Has HSE reviewed whether the guidance that you have on surface transmission remains appropriate?

Professor Curran: I would have to check with my policy colleagues, but, certainly, I have regular conversations with all of my—

Q2540 **Chair:** You are the chief scientific adviser. You would know, I am sure.

Professor Curran: Exactly. I have regular conversations with my policy colleagues. We have regular meetings where we review those new evidence statements, but there has not been, as I say, the requirement based on the evidence that we have been using—ie the evidence from SAGE—to suggest that we would need to reduce the advice that we are giving on surface contamination, because I just do not think the evidence says you can rule it out. If you cannot rule it out, I do not think it is appropriate to say you should stop cleaning regimes to the level that are currently in place, because that would be based on very, very limited evidence, and, of course, I do not think that would be appropriate.

Q2541 **Chair:** Is that the normal standard of the HSE—that you have to take action if you cannot rule a risk out?

Professor Curran: The information on Covid is slightly different. We are dealing with a national pandemic, are we not? It is really important that we consider what are the potential routes of exposure for this particular issue. When we are looking at all other risks, obviously, we take the whole situation into account. We take our evidence from the practical side of work, we take it from the experimental side of work, and that determines the guidance we would provide in a standard way.

Q2542 **Chair:** I would be very grateful if, after this hearing, you would write to us with a summary of what formal evaluations you have made of the requirement to continue the same guidance that has been in place since near the beginning of the pandemic.

Professor Curran: I am happy to do that.

Q2543 **Chair:** We are very grateful to you. We have overrun our appointment with you slightly, but that is because it has been such an interesting area. I thank you, Professor Curran, and Professor Mondelli for joining us from

Italy, and giving us the benefit of your expertise. We are very grateful for that.

Professor Mondelli: It was a pleasure. Thanks.

Q2544 **Chair:** Thank you very much indeed.

Examination of witnesses

Witnesses: Professor Pollard and Dr Alroy-Preis.

Q2545 **Chair:** We will now go to our second panel of witnesses. I welcome two very expert witnesses to discuss vaccines. We are very grateful to have back with us Professor Sir Andrew Pollard, professor of paediatric infection and immunology at the University of Oxford, and, as we all know, the chief trial investigator of the Oxford-AstraZeneca vaccine trial. Thank you very much indeed for helping the Committee again today. Joining us from Israel is Dr Sharon Alroy-Preis, who is director of public health services in Israel. Thank you very much indeed for joining us and informing our Committee today.

I will start with a couple of questions to Sir Andrew. When you were last with us in June of this year and looking forward to what we know about the likely development of Covid, you said that the likely course of the pandemic was that new variants would become more transmissible, but the likelihood was that vaccines would continue to protect against severe illness. Does that remain your view?

Professor Pollard: It does remain my view, and that is the case. One of the difficulties that we have in all of these discussions at the moment is the rigorous understanding of the data. Rebecca Long Bailey commented about the very high case rates in the UK, which, of course, is true, but, actually, I am sure you are referring to cases, which is very much related to the amount of testing. If you look across Western Europe, we have about 10 times more tests done each day, per head of population, than some other countries. We have to adjust always by looking at the data based on the rates. We have a lot of transmission at the moment, but it is not right to say that those rates are telling us something that we can compare internationally.

Interestingly, Israel also has a very high testing rate and picks up a lot more cases than many other countries. A lot of our policy decisions should be very much focused on what we think is right for this country, not by saying other countries have much fewer, because it is very difficult to make those assessments. If you make the adjustment of cases in relation to the rates of testing and look at test positivity, currently, Germany has the highest test positivity rate in Europe.

When we look at these data, it is really important not to bash the UK with our very high case rate because it is partly related to our very high testing rate. I am not trying to deny that there is plenty of transmission

at the moment because there is. It is just that the comparisons are problematic.

Q2546 **Chair:** Thank you. That is extremely helpful. To take that further, when you were with us in June, you warned that, irrespective of the level of testing, we should not be over-focused on positive tests because the likely course was, as you thought at the time, that more people would contract Covid and therefore we could unnecessarily worry about levels of infection, which, if things worked out as you anticipated, would rise, but that the key test of how alarmed we should be is severity of disease and hospitalisation. Is that right?

Professor Pollard: That is absolutely right. We now have to look at also the rigour of the data when we look at hospitalisations and deaths. Let us take some of those different segments. For example, the vast majority of intensive care admissions remain the unvaccinated.

On the whole question about booster doses and so on, and whether the vaccines are failing, in terms of severe disease, we remain in the situation that the way to control that is for those unvaccinated people to be vaccinated. You could argue that all the other measures—having more people boosted, mask wearing and so on—will also have an impact, because of course it will do, on those unvaccinated individuals.

If we want to protect intensive care, there are measures to do that, as we did last year, with non-pharmaceutical interventions, but we could also be vaccinating those individuals and focusing on that bit, which would protect intensive care.

If you look at hospital admissions, which is a slightly different measure, generally speaking, that is quite a different story from last year. If you remember, last year, with no vaccine programme, hospital admissions were a relatively serious consequence of Covid infection with severe lower respiratory lung disease and, sadly, many people going into intensive care and dying overwhelmingly in intensive care units, whereas today, although there are some people whose immunity has flagged, the vast majority of people going in are not following that same course. The hospital length of stay is much shorter. Those individuals who are going in have a much milder disease. Interestingly, many of them have other underlying health conditions that have destabilised by having a relatively mild Covid infection. Physicians see that every winter with other viruses. People who are frail with various health conditions will be tipped over the edge as a result of those viral infections. Covid is doing that as well. If we put further downward pressure on Covid those admissions would stop.

Partly here, we have to think about the long term. Are we trying to protect the NHS by what we are discussing today? That is probably the main question that is being raised, certainly, in the media and in Parliament at the moment.

When you look at where the NHS is today, it is incredibly fragile, whether it is in primary care, secondary care or social care. That fragility is only contributed to a small amount by Covid. Vaccinating is not going to suddenly make the NHS okay and not be on its knees where it is at the moment. Certainly, the pandemic has had a major impact on that with waiting lists and so on.

Can vaccination make a difference? Certainly, more vaccines for the unvaccinated will make a big difference to intensive care, and booster doses for those who are frail will likely reduce some of the admissions, but it still does not change the overall needle on where we are with a very stressed NHS.

Q2547 **Chair:** We have embarked on a booster programme. The reason for that for most people other than those with diminished immune systems is because of waning protection of the vaccines. Am I right in thinking that one has to deconstruct waning, as it were? There are two aspects of waning protection—one is from contracting Covid and the other is from severity of disease. Sometimes they are conflated. Is it right to draw a distinction?

Professor Pollard: It is absolutely right to draw a distinction. If we look at cases in the community, because of the escape of the virus from neutralising antibodies—antibodies are important for controlling infection per se—we have seen, despite a vaccinated population, transmission in that community. It does not mean that the vaccines are not working at all because transmission is much lower than it would be in an unvaccinated population with the amount of social contacts we have. The virus is evolving all the time. There is a new variant, as you are aware, that may take off and is a bit better at transmitting and, therefore, escaping those immune mechanisms. That is our long-term future with this virus.

If we boost, we will have much higher levels of antibodies, at least in the short term, which will help to control that. Boosting will get those numbers down and make us feel better that we are doing better than other countries if we are in that competitive world, but the key question in my mind is still what is happening with severe disease. The severe disease is the intensive care unit admissions. You can reduce those if you have less transmission because we are protecting people by there being less virus out there. But, in the end, the unvaccinated people will meet the virus and, if they are not vaccinated, those susceptible will end up in ICU. It just might not be today; it might be next year. That is why we still have to focus on persuading those individuals who are unvaccinated to be vaccinated. That transmission that we are talking about will still cause those milder hospital cases that are in those frailer individuals, and boosting can reduce their risk of that happening.

Q2548 **Chair:** Finally from me on this, given what you have said, does it give you any concern that for perfectly understandable reasons we have become familiar with looking at the daily figures on new transmissions

and the charts that are put up every night on the news, and comparing them? The graph goes right back to the beginning of the pandemic, but we ought not to be as concerned today about any given level of transmission as we were in the past. Do we run the risk of being worried about the wrong thing?

Professor Pollard: It comes back to the rigour of understanding the data we look at. It is presented in that simple way because that is the only way you really could do it in real time. There is not time to do the detailed analysis of what those figures mean. It is important to remember if we look at deaths that that is deaths within 28 days of a positive test. If you have a lot of transmission in the community, lots of people will die from lots of other causes that are not Covid but will be included in the numbers. If you look at the death certificate data or the more detailed analysis that is done later, you will see much lower Covid death rates because lots of people have died having recently had a positive test but did not die from Covid. The death rates are quite misleading at a time of high Covid in the community.

Secondly, the hospital admissions data are also misleading because they are also generated in real time; there isn't the coding that happens in the NHS, which takes quite some weeks after the hospital admission to work out what the person was admitted for. If I am admitted for appendicitis today and I had a Covid-positive test, that will appear in the daily data. If you have lots of transmission in the community, the hospital admission data that we look at each day will reflect the fact that, whatever you go into hospital with, a proportion of people will have been positive in the past month. We really have to look in more granular detail at the formal analyses that are done rather than looking at just the raw data, which are quite misleading. It is important to emphasise that that does not mean that there is not Covid transmission and people being hospitalised with it. It is just that the rigour of how we look at the data needs to be addressed here.

Q2549 **Chair:** Do you have any feel for, or any lens into, what the proportions are of people who are recorded as having died within 28 days of a positive Covid test—people who died of Covid rather than died of something that they were admitted to hospital for alongside Covid?

Professor Pollard: I do not have those numbers. We need to have some formal look at the data, and you have to link the two datasets together to be sure that you are looking at the same groups of individuals to give that answer. I do not have that.

When you look in the community, for example, we see high rates of transmission. In some parts of the country, the vast majority of those come from very effective testing in schools, so we are picking up a lot of very mild infections. We know from all of the previous studies done that children contribute a relatively small amount to adult transmission. Of that very high number in some regions, a proportion of cases reflects

something that is transmission among children, which is of much less importance than transmission to older adults.

Q2550 **Chair:** Thank you very much indeed.

Dr Alroy-Preis, Israel has been much in the news over the last two years and much studied. You were very early to vaccinate the population. You have experienced a rise in the level of infections recently and brought in a booster programme. Explain this to us. Does it confirm what Sir Andrew has been describing—that it is people who are being infected but not necessarily severely ill?

Dr Alroy-Preis: Thank you for inviting me. I have to start by saying that every country has the obligation to make its own decision based on its own setting, and I am not here to say what the UK needs to do but just to explain what happened in Israel. What we saw in Israel is that we got to a high coverage of the population in around March 2021, which is about 60% of the population covered. We have a young population. About a third of our population was not eligible for vaccination, and still we got to 60% coverage. In June, we started to see daily cases rising by more than a hundredfold in a month and a half, from about 10 confirmed cases a day to more than 1,000.

What was more worrisome is your question. We saw the same rise in severe cases. We had a more than tenfold rise in severe cases in a month, and 60% of them were doubly vaccinated. That was an alarming signal for us. When we looked at the data, we saw that there was twice or three times more risk for people who were vaccinated early on in January compared to those who were vaccinated later on in March and April. It was clear that at least part of the picture was waning immunity.

Another thing that helped us was looking at the UK data and seeing that the vaccine effectiveness in the UK remained high around 88% from what was reported, whereas in Israel the vaccine effectiveness dropped at the beginning to 60% and then 40%. At the beginning it was just for confirmed cases, but the severe cases effectiveness remained high. As time passed, vaccine effectiveness reduced also for severe cases.

In addition to our vaccine effectiveness among 12 to 15-year-olds, teenagers who had fresh vaccine were approved for the vaccine in June. When we got to this rise in cases in July, they were still in the timeframe when they did not have waning immunity. For them, we saw vaccine effectiveness of around 90%. We knew it was not the issue of the Delta itself. It was not the variant that was causing most of the problem, although it contributed to some extent because with the Alpha variant we saw vaccine effectiveness of 95% to 96%, so there was some drop, but the main surge in cases was due to waning immunity. This led us to go to a booster campaign, starting with the older age groups and then going down by steps.

Q2551 **Chair:** Thank you. Just briefly, give us a thumbnail sketch of the impact

of that as it is discernible already.

Dr Alroy-Preis: On a personal level, you see that someone who gets a booster dose and has a fresh vaccine is more than 10 times protected against severe disease—any confirmed cases but also severe disease, especially in those who are 40 and above. But on a national level what we saw was a drop in the surge of cases. Before we started the booster campaign, we had a reproductive number of 1.3 to 1.4 for weeks. It was a really high number, causing us to double our confirmed cases every week or every 10 days. This is how we got from about 10 cases a day to more than 1,000. Once we started the booster campaign, the reproductive number went down. Now, it is between 0.7 and 0.8, so the pandemic surge is now shrinking. We are seeing a drop in cases, obviously in the vaccinated individuals, but also in the non-vaccinated. We are getting to the point where the vaccinated individuals are also protecting those who cannot be vaccinated.

Chair: Thank you very much indeed.

Q2552 **Dehenna Davison:** Dr Alroy-Preis, I will stick with you if that is okay. Thank you for joining us today. I am keen to get a proper understanding of the decision-making process and the research that was done behind the introduction of the booster vaccines. The world has been watching very closely what has been going on in Israel given your pioneering nature throughout Covid. What research was actually conducted into the waning immunity? Was that particularly waning immunity around infections, or were you seeing that manifest in hospitals as well?

Dr Alroy-Preis: We saw it in everything. In the beginning, we were seeing this in confirmed cases. Since the beginning of the vaccination campaign, in our Ministry of Health, we were following the vaccine effectiveness since January, and saw a really high level of vaccine effectiveness once we got the vaccine going. It was around 95%. In the beginning of our fourth wave, we saw a drop, first to 60%, still remaining very high, 95% for severe cases and mortality, but as time went by we saw a drop in that too.

We knew that Israel was the first to vaccinate. I think we were about three months ahead of the rest of the world in our vaccine campaign. We reached a high level of coverage in March, whereas in the UK you reached it around June. There is a three-month period where we can see the effect of waning before other countries will. Once we saw that, we brought together a group of scientists from four leading academic institutions in Israel—I can tell you their names, but I am not sure it would tell you anything—and tried to see if different scientists looking at the data by different statistical methods will get to the same result to make sure that we are not making any type of mistake. There was a really unique collaboration of those four institutions, making sure that we were seeing what we were seeing. What we saw was waning immunity, first, for confirmed cases, but also for severe disease and mortality in the ages of 60 and above. Now, with time going by, we were also able to

show a statistically significant difference for 40 and above in severe disease and mortality.

Q2553 **Dehenna Davison:** How was the decision made about the point at which to implement the booster vaccines? There is a debate going on in the UK at the moment around whether six months is most effective or whether we reduce that to five months. How was that decision made in Israel?

Dr Alroy-Preis: In Israel, we have, very much like the FDA, a vaccine advisory committee that is advising the Ministry of Health. Several professionals—infectious disease epidemiologists, lab scientists and others—are there voluntarily giving their advice, and the data was shared with them repeatedly. We brought it to the advisory committee three times before we all felt really safe. They vote. In the first two rounds of voting, most of the people voted that a booster is not recommended yet. We saw the same thing. There were confirmed infections but the severe disease is not impacted by that.

As more and more data was gathered, and we saw the effect on severe disease, the advisory committee voted for a booster dose, first for those who were 60 and above and healthcare workers, and then expanded it to other age groups as time went on.

We have a process in Israel of a committee of about 60 to 70 professionals in different sectors advising the Ministry of Health. The CEO or the director of the Ministry of Health actually makes the decision based on their recommendation.

Q2554 **Dehenna Davison:** What is the length of time between the initial vaccination programme and the booster period, and was that quite heavily debated?

Dr Alroy-Preis: We started seeing the rise in cases around mid-June. We started the booster campaign for 60 and above at the end of July. We saw an exponential rise in severe cases, so we had to act quickly. Based on our predictions, if we had not done the booster and remained at a reproductive number of 1.3 to 1.4, with the numbers rising and doubling every week or 10 days, we would have got to 1,500 severe cases in our hospitals at the end of August. This was beyond the ability of our hospitals to take care of them, so we had to move really quickly. As I said, there were at least two meetings where people did not feel that there was enough data to suggest it, and only when we saw a real impact on severe cases was there an approval to go ahead with the booster dose for the elderly.

Q2555 **Dehenna Davison:** Have you found that the take-up of the booster doses has been similar to the initial vaccination programme, or have you seen people dropping off?

Dr Alroy-Preis: It is different between the older age groups and the younger age groups. With the older age groups the compliance was very high, and it was very similar to the first wave. With younger age groups,

there was some hesitancy among people feeling that maybe they are not at risk. As we saw more and more cases of people who were 40, 45 or 50, with one chronic disease or not at all and doubly vaccinated getting into severe and critical conditions in the hospital, the compliance went up. We are above 50% compliance even in those age groups.

Another thing that is helping that—it is not the purpose of it—is the green pass. As in Italy, we have a green pass in Israel. Once the disease surge went up, we implemented a green pass where you need to show either a vaccination certificate or recovery certificate, or do a negative test. That is also leading younger individuals who do not feel at risk to be vaccinated with a booster dose.

Dehenna Davison: Thank you, Dr Alroy-Preis. If I have time, Chair, could I put a question to Sir Andrew as well?

Chair: Yes, sure.

Q2556 **Dehenna Davison:** You mentioned that you think that one of the huge risks is the unvaccinated population and those presenting in hospital with severe illness. At the moment the Government have an advertising campaign to encourage people to take up the booster jab. Do you think the Government should be doing more to encourage people to take up the initial first and second dose?

Professor Pollard: As our colleagues in Israel have shown, there is no doubt that if you use a booster you will get a big increase in immune responses. We have good data across all the vaccines that we use here to show that. That will have further downward pressure on the virus just like all of the other things such as mask wearing.

In respect of the question of whether the booster programme could have a protective effect for the unvaccinated, the answer is yes, but my case here really is that, in the long run, if we take a long-term view, those individuals remain at risk. The virus is not going to completely disappear, so the unvaccinated still need to be vaccinated. We need to do all we can to encourage them to be vaccinated as well.

Dehenna Davison: Great. Thank you.

Q2557 **Chair:** Thank you very much indeed, Dehenna. I will bring in Catherine in a second and then Carol. Sir Andrew, in terms of the effectiveness of the vaccines—perhaps the one that you are responsible for—what is the difference between its effectiveness against transmission and against severe disease? What are the relevant percentages?

Professor Pollard: We are seeing much lower protection with Delta against infection. The question about transmission is much murkier, but if you look at infection in the community—

Chair: Yes, infection.

Professor Pollard: —we see much lower rates of effectiveness there. I think that was also very clearly shown in Israel where the dominant vaccine used is the Pfizer vaccine.

Q2558 **Chair:** What sort of percentage are we talking about?

Professor Pollard: It depends on which study you look at, but somewhere around 50% or below 50%. In some of the studies, it is around 40% for the vaccines that are currently being used. If you are looking at what is likely to happen in the future, the virus will evolve to be able to transmit even better. I would expect that number to be going down over time because we know that it is the neutralising antibodies the virus is escaping from, and that will result in a lower measure of effectiveness. The key question to me remains the severe disease one.

Chair: Indeed.

Professor Pollard: The severe disease figure stayed extremely high. If you look at the best analyses that are being done, we are still somewhere between 80% and 95% in the UK against severe disease. The difficulty is doing these analyses in populations with very high vaccine coverage. To rehearse that a little bit, essentially what you are trying to do is to see the rate of severe disease in those who are vaccinated versus those who are not vaccinated. For those who are vaccinated and have had two doses, there are much higher rates in the elderly than in younger age groups. For the unvaccinated, a high proportion of those—now an increasing proportion—have been infected. We know that infection will also give you protection. You are trying to say how effective the vaccine is in relation to a control population who—

Chair: Who is diminishing.

Professor Pollard: They are diminishing, but many of them are already immune, so they are no longer true, unprotected individuals because they have had an infection. It becomes increasingly difficult to measure what is changing over time in effectiveness.

We then have another problem. If you want to know what the effectiveness is in an 80-year-old, most of the studies that are done do not have enough unvaccinated 80-year-olds to do that comparison, so you have to compare with a younger age group, who, obviously, have a completely different set of risks from an 80-year-old. We have great difficulty at this moment in being certain about effectiveness figures because they are so confounded by the remarkable programmes that we have that protect so many people and, of course, the infection in the community.

We have very high levels, but I do not think, when you look at any of these numbers, that they are absolute truth because they are really difficult studies to do.

Chair: Thank you very much. Katherine Fletcher.

Q2559 **Katherine Fletcher:** Thank you, Chair. Sorry, I thought erroneously that you might have forgotten me.

Dr Alroy-Preis—I hope I said that right—thank you so much for your time. It is hugely appreciated that you are giving your knowledge across the oceans, as it were. I want to return to the idea of waning of immunity and understand the root cause of it. You have been quite clear that a variant in the Covid virus itself is unlikely to have been the cause of the waning immunity. Have you had any opportunity to work out whether different vaccine types have different rates of waning and also whether it was the dosage interval that was delivered in Israel that perhaps might cause it? To help us learn some lessons in the UK, can you talk to those two points?

Dr Alroy-Preis: Those are great questions. We do not have much information in Israel from different vaccines. The main vaccine we have used has been Pfizer, especially early on. Now, we are using other vaccines, but early on we had mainly Pfizer vaccines to give, so that is what most of the population got. There is a high amount of research on that topic from across the world showing waning for other types of vaccines as well, whether it is Moderna, AstraZeneca or Johnson & Johnson. There is a lot of data showing that there is waning.

Whether part of the waning is because of the FDA-approved protocol that we used of three weeks apart between the first and second dose is a really good question that I do not have an answer to, because part of the difference between Israel and the UK is also the timeframe. If we are three months ahead, we will see the waning three months before you see it. I am not sure if part of what we are seeing is because of the protocol that was three weeks apart versus 12 weeks apart or because we are seeing the waning first. Maybe with time, we will have the answer to that.

On the point about who we are comparing to and are we basically comparing our vaccine effectiveness to those who have recovered—we know that they are protected; we have data to show this also, because we are following the recovered and their rate of recurrence of disease—it really is a challenge.

Q2560 **Katherine Fletcher:** Fair play. Thank you. Professor Sir Pollard, are we doing anything with our broader spread of vaccination types to track relative effectiveness?

Professor Pollard: I think so. It is a really important question. I completely agree with the difficulties in making these comparisons except in countries where you have done things exactly the same. Of course, we have not done that here either in that we have used the vaccines in slightly different populations. If you see differences between them, it may just be that there are differences between the populations that you have vaccinated rather than differences in waning.

What we have in the analyses being done in the UK at the moment is quite a confusing picture. The ONS study from about a month ago

suggested that Pfizer was waning in protection against mild infection more rapidly than AstraZeneca. The Public Health England study suggests that AstraZeneca is waning more rapidly than Pfizer. Working out the truth is difficult.

The important point is that, if there is some waning of protection, it is still relatively minor at the moment, and it is not suddenly falling off a cliff. A point I have made before is that we would not anticipate the protection to suddenly fall of a cliff. It may decline somewhat, but certainly with severe disease it just does not make biological sense for that to happen.

The US has some data looking at waning that suggests that Moderna may be slightly better than Pfizer in producing stronger, more long-lasting immune responses. All of these are slightly difficult to interpret. If you go to the hospitals, the intensive care units in this country—I accept that Israel may be different—are full of, largely, the unvaccinated. On the wards, we have hospitalisations, but they tend to be people with other health conditions who are a subset of the population who may not have responded so well and may wane more quickly.

Q2561 **Katherine Fletcher:** That is really helpful. In essence, we are almost at the point with this pandemic where we are down to personalised medicine and the variate factors that might make it a much longer study. But the clear message is that, if you have not been jabbed, at some point if you are at risk, you could end up in an intensive care ward.

Professor Pollard: That is the absolute key. If you have been vaccinated, even in the context of waning, you are in a very much better position than anyone who is unvaccinated.

Katherine Fletcher: Fair enough. Thank you.

Professor Pollard: But the boosters that are being rolled out at the moment are going to have an additional impact on that because it will raise the immune response as well.

Katherine Fletcher: Thank you.

Chair: Thank you very much.

Q2562 **Carol Monaghan:** Professor Pollard, why Pfizer for the boosters?

Professor Pollard: I was not party to the discussions about which vaccine to use. My guess is that when you look at the strength of the immune response—this is not about protection because we know people are highly protected—the strongest immune responses are likely to be with the RNA vaccines, so that is probably the rationale. There will also be other questions about what is available and what the supply is and so on. I am not party to those discussions.

Q2563 **Carol Monaghan:** Okay, thank you. Dr Alroy-Preis, you have talked about your vaccination programme for children. First of all, are you giving

your 12 to 17-year-olds two doses?

Dr Alroy-Preis: Yes.

Q2564 **Carol Monaghan:** Okay. What impact has that had on infection numbers?

Dr Alroy-Preis: The beginning of the vaccination programme for the 12 to 15-year-olds was when we were between waves, so the compliance was not very high. Once the fourth wave started, the compliance went up, and we have about 60% coverage for that age group now. We saw a reduction by about a half in the number of cases, and to some extent even more than that—the underestimation is half. It has an impact on confirmed cases for that age group. We all know that for 12 to 15-year-olds the risk of severe disease is really low if you are a healthy young child or teenager. We could not see an impact on hospitalisation, obviously, but the vaccine effectiveness for that age group was around 90% in preventing disease.

Q2565 **Carol Monaghan:** Did you see any impact after the first dose, because in the UK we are only doing one dose at the moment?

Dr Alroy-Preis: We saw that one dose gives a vaccine effectiveness of about 40% to 50%. This is the same number that we saw in our first vaccination campaign. We are looking at the vaccine effectiveness of full protocol versus someone who is midway and the rate of disease among those individuals. It is roughly 40% to 50% for one dose. We are recommending for everyone to get a full protocol, which means a first dose and after three weeks another dose.

Q2566 **Carol Monaghan:** One of the concerns about the vaccination of young people is the instances of myocarditis. Have you seen any instances of that in Israel?

Dr Alroy-Preis: I will start with the first vaccination campaign, which was for 16 and above. Among more than 3 million people vaccinated, we saw 142 cases of myocarditis. We published this after seeing that there was a connection to the vaccine mostly in males aged 16 to 30, and mostly after the second dose of the vaccine in the first week after the vaccine. Ninety-five per cent. of the cases were mild and discharged without any sequelae. In another piece of research done by one of our biggest HMOs, Clalit, which followed 2 million people and looked at the data after discharge—the Ministry of Health does not have the follow-up data—in all of their cases there was full recovery even when they looked at imaging studies like echo and other types of data that they had.

We are concluding from that that, yes, there is a risk of myocarditis, especially in the 16 to 30-year-olds, especially in males, especially after the second dose, but in most cases that risk is of mild disease, and so we are still recommending that. When we rolled it out to 12 to 15-year-olds, we did not see the same rate; we saw a lower rate of myocarditis in that age group.

Q2567 **Carol Monaghan:** I was going to ask if there was any consideration of giving the 12 to 15-year-olds a single dose, but if you are saying you did not have the same rate of myocarditis was that one of the considerations when you were giving the second dose?

Dr Alroy-Preis: That was one of the considerations. The second was that the clinical manifestation of the myocarditis was mild in most cases. There is a risk. The event is rare—it is not common—but even when it happens it is a mild disease. Looking at the data from the research done by Clalit, the risk of myocarditis from Covid-19 is higher when you have the disease than when you have the vaccination. If you are dealing with a disease surge and you have many cases, and the risk of you contracting Covid-19 is high, the risk of myocarditis as a side effect from the disease itself is higher than from the vaccine. That is why during the fourth wave, when we had 10,000 cases a day for several days, we encouraged teenagers aged 12 to 15 to take the vaccine, knowing that even if you have this adverse event it is mild for the most part. Once it was rolled out, we saw that the events were at a lower rate in that age group.

Q2568 **Carol Monaghan:** Thank you. Professor Pollard, the UK has only given a single dose to 12 to 17-year-olds. What do you consider the impact might be in hospitalisations and severe infections?

Professor Pollard: The second dose, as I understand it, is still under consideration. It is not that it is only going to be a single dose. It is just that the decision has not been made yet.

The other important point is that we have already had a lot of infections in teenagers, which essentially, for priming their immune system, will be like having had a first dose. For a lot of people, that single dose is like having a second dose, but, of course, there is still a proportion of individuals who have not had a first or second dose. If we want to reduce both the risk of infection and the very, very rare chance of severe disease in children, certainly getting the first dose will have an impact on that. It may be that second doses will be decided on at some point in the future.

Q2569 **Carol Monaghan:** Okay. So, you would know more about the considerations that have been taken.

Professor Pollard: Yes.

Q2570 **Carol Monaghan:** It would be things like those we have heard already.

Professor Pollard: The main issue here is to fully assess the myocarditis data to make a decision on the second dose.

Q2571 **Carol Monaghan:** How soon do you think that decision will happen?

Professor Pollard: I am not involved in the discussion of when they will make those decisions.

Carol Monaghan: Thank you, Chair.

Q2572 **Chair:** Thank you very much indeed, Carol.

Obviously, it is tremendously good news, and we ought to pause to reflect on the fact that we are discussing a context of continuing high efficacy against severe disease from the vaccines that we have available. There are some groups for whom that is not true. People with blood cancers, for example, are finding that the vaccines are not providing the protection that other groups have. In your work at Oxford, do you have any insight into what interventions might be available, other than shielding, that might offer some hope for such groups?

Professor Pollard: The first thing to say is that we now have a policy to select those individuals who are immunocompromised—not just the blood cancers but the wider group—to have what has been called a third dose. That is because of the observation that there are some individuals who make very little response with the first two doses but giving them another gets them started up. That programme will help some individuals. What is not clear at the moment is how long being souped up lasts for if you are one of those individuals. There are still some who will not respond to vaccines, and that is many different conditions, not just the blood cancers. For those individuals, there are essentially two approaches. One is the use of monoclonal antibodies, which have recently started becoming available, and the second is the use of antiviral drugs. I have not been working on those areas, but both of those provide potential additional benefits for those individuals who cannot respond to vaccines.

Q2573 **Chair:** We should put on record, as we highlighted in our report, the role of treatments, which has developed considerably thanks to research efforts.

Going back to what you were saying at the beginning, Sir Andrew, about the level of testing being higher in this country than in other countries and this giving a higher number of cases, you said that a lot of that is in schools. Given that, in schools, we have heard both from Israel and here that young people tend to have a very mild experience of Covid if they get it, and you yourself have said today and before that having Covid provides the equivalent of a dose of one of the vaccines, are we being too cautious in having such a rigorous testing regime in schools? In schools at least, should we not be a bit more relaxed about transmission?

Professor Pollard: The first thing is it is absolutely critical that we keep children in schools. We have seen that the biggest impact of the pandemic on children has been missing school and the psychological effects of that. I really think all of the policy decisions should be focused on that.

Clearly, the large amount of testing in schools is very disruptive to the system, whether that is the individual child, who is then isolating because they have tested positive but they are completely well, or it is because of the concerns that that raises more widely in the school. We are aware of families taking their children out because someone has tested positive in a school. There is a huge impact of widespread testing in schools.

We probably need to move in the pandemic over this winter, or maybe towards the end of the winter, to a completely different system of clinically driven testing—in other words, testing people who are unwell rather than having regular testing of those people who are well, because that drives a lot of these actions that happen particularly in schools if you are having lots of asymptomatic testing. If someone is unwell, as should normally happen, they should not be going to school, but that is not a Covid-specific issue. It should be a general rule that if someone has the flu they should be at home and not at school.

Q2574 **Chair:** You say "in the course of this winter". You are clearly describing a move towards "in the long term", which is consistent with your view that in the long term Covid is going to be in circulation, and if we are vaccinated the vast majority of people will get it in a mild form rather than a severe form. If one were to move towards that, given the mildness of the experience for children, would schools not be the best place to start that transition, as it were?

Professor Pollard: I think it is a good place to start that transition. If you remember, when the JCVI looked at vaccinating adolescents, their view was that there were likely to be marginal benefits in doing so overall in the population, so they came down with a view not to. It was after the review by the chief medical officers that the decision was to roll out vaccination. Because the benefits from a Covid perspective are marginal, it is reasonable to take either view—to either vaccinate or not to vaccinate. The reason for the vaccine programme that the chief medical officers had was to keep children in school, so we have to absolutely do that as part of that contract with families that their children are being vaccinated because we want them to be staying in school.

Q2575 **Chair:** As to the implications for the testing programme, one thing that our joint inquiry was critical of in the test and trace programme was not to anticipate what was ahead in terms of the demands. Obviously, any use of public money is at the expense of some other use of public money. Should we be preparing ourselves in the medium term for a lower level of testing of asymptomatic people and gearing ourselves up for a different model of testing?

Professor Pollard: That is an inevitable future anyway. We are not going to be testing at this rate for Covid forever, so we need to think about how that transition works. As I have said, there clearly is a lot more transmission at the moment, and that adds some additional pressures on the NHS because there are some individuals going into hospital, and more than there were before.

I think we are in an improving situation because of the high vaccination coverage that we have. The booster programme will start limiting even further the number of cases, and particularly those hospitalised cases. There is lots of infection happening in the community that is boosting lots of the younger people as well, including children. All of that is an improving situation with higher levels of population immunity, which

means that at some point we will reach a more steady state with this virus and that is likely to be more manageable. The problem is that we do not know exactly when that is. There may still be some surprises around the corner. So there is uncertainty.

Q2576 **Chair:** When you have appeared before the Committee before and in other public fora, on boosters you have worried not that boosters will be effective and useful but that they may be more useful applied to other countries that have not had first doses. Does that continue to be your view? Would you prefer that we were sending them to places to protect people for the first time?

Professor Pollard: We are still in that window where there are many countries with very low rates of vaccination, particularly in Africa, but it is not solely in Africa. For individuals in those risk groups—older adults and those with other health conditions—a dosing that could be given to those individuals has a much greater benefit than a booster dose to any of us. From a global equity point of view, there is still a strong argument for more actively sharing doses. Obviously, today, we have been talking about the domestic situation—

Chair: Indeed.

Professor Pollard: —and from a domestic perspective there are some additional benefits, but they are much more marginal than the benefits of vaccinating someone who is completely unvaccinated and is an older adult. That, I hope, is an improving situation over the next six months, but it very much depends on what happens with supply. The whole issue around supply is that, if the whole world decides to start boosting now, the countries that were behind will be left even further behind. So there is that issue.

One other thing to pick up on that was talked about earlier is the timing of the boosters, which is a really important question. There is no doubt that if you boost, as Israel did, at almost any point you are going to boost immune responses and have better protection. If you take a long-term view, you would boost as late as possible. That is why six months has been considered. This is based on immunological principles. This is not a great dataset that says for Covid vaccines and each individual one of them when the right time to boost is. Generally speaking, if you leave enough time for the immune response to mature and for the previous antibody levels to have dropped a bit, you get a better boost.

The six-month figure—with some vaccines, we do it at a year or even at later points—means that you get a stronger impact. The question is whether you have enough of a problem to bring forward the doses. Are we facing a disastrous winter and we need to boost everyone earlier? That would be completely rational if that is the case. We need the modellers to help us with that. If we are taking a long-term view that we want to have protection over the next year or even more than that, having the booster later from an immunological point of view is better.

The policy is more difficult because you have to look at what is happening now, but the long-term view would be to boost later.

Q2577 **Chair:** Thank you. I see that. To bring it full circle, do you think that there is a danger that high levels of infection reported panic us into bringing forward a booster programme or the interval, which is at the expense of longer-term protection for reasons that go beyond what is clinically necessary?

Professor Pollard: I think it is highly unlikely that boosting at five and a half months or six and a half months makes any difference. I do not think what is being proposed is problematic one way or the other. If we were thinking of boosting people much earlier, that would be unwise, and it is also not needed.

Q2578 **Chair:** Is there any advantage in boosting much later, say, a year?

Professor Pollard: We do not have the data for the Covid vaccines to say what difference in magnitude or duration would be achieved. Six months is a good compromise. That is what we have done with many vaccines in the past, and we see very good responses. I see Sharon has her hand up.

Q2579 **Chair:** Dr Alroy-Preis?

Dr Alroy-Preis: I know there are different approaches to kids' infections because the act of disease, when it happens, is mainly mild or asymptomatic, but part of what is influencing our decision making is the effect of long Covid. When we looked at that—and I also saw data from the UK supporting this—one in seven or eight kids has continuous symptoms after recovery. We saw the same thing in the research that we did among 14,000 parents of recovered kids. We saw in the 12 to 18 age group that around 5% of the kids, even six months after recovery, were still having symptoms from long Covid. That is one thing.

The other thing is PIMS, the multisystem inflammatory response that happens. We think that it is happening more in Delta than we saw before. We are looking at the data right now. That is another risk.

The third risk is the unknown. We have many viruses that can create complications years after an asymptomatic infection to begin with. We are looking very cautiously at infections in kids. Kids that are still coming in contact with a confirmed case need isolation. We now have a programme of a green class trying to get them back in school, because I am completely in agreement that we want kids in school as much as possible. However, we are trying to make it safe for them and in the settings in which we have more infection spread among those green classes we are trying to stop them or prevent them from happening, but for the 80% that do not have spread of infection when you have a confirmed case in the school we are trying at least to allow those kids to learn with frontal learning.

Our approach is trying to lower the number of cases. Once the number of confirmed cases comes down, we have less risk for kids, less isolation and more kids in school. It is challenging with the surge of disease. Even though we are performing 80,000 tests a day, we are seeing a drop in the percentage of positive test results. We are remaining very high on testing and seeing a drop in the infection rate. This is what we are trying to aim for. I know that there are different approaches. With the uncertainty that is happening here, probably every approach can be justified, but this is the Israeli approach.

Chair: Dr Alroy-Preis, we are very grateful for having that directly from you. We have said in the report we have issued that it is very important that we should look at what other countries are doing, grappling with a virus that affects the whole world. To have your direct testimony is a real privilege. We are very grateful for that.

We are grateful, as ever, to you, Sir Andrew, for helping the Committee think through the next steps. You have made a signal contribution to getting us to where we are today, and we are very grateful for your continued guidance. Thank you both for appearing today.

Dr Alroy-Preis: Thank you.

Examination of witnesses

Witnesses: Professor Holgate, Professor Chappell and Dr Waite.

Q2580 **Chair:** We move now to our third and final set of witnesses. I will introduce them as they are taking their seats at the table here in the Committee room.

We are going to discuss preparations and prospects for this winter. To help us do that, I am delighted to welcome Professor Sir Stephen Holgate. He is professor of immunopharmacology at the University of Southampton and chair of the Academy of Medical Sciences' expert advisory group, which, at the request of the Government's chief scientific adviser, carried out reports last year and this year into preparing for winter. Sir Stephen, thank you very much for joining us today. I am very pleased also to welcome Professor Lucy Chappell, who was quite recently appointed chief scientific adviser at the Department of Health and Social Care, and Dr Thomas Waite, whom we welcome back to the Committee. Dr Waite has helped us before. Since the beginning of July he has been interim deputy chief medical officer. Thank you all very much indeed for appearing.

To start with Sir Stephen, perhaps you would summarise the advice you gave, on behalf of your colleagues at the Academy of Medical Sciences, to Sir Patrick and therefore the nation about what we should be doing to prepare for this winter.

Professor Holgate: Thank you very much for inviting me to speak to you. This was the second report. We had already experienced during the

first report whether or not any of the predictions that we came up with in that first report panned out over the following 12 months. As it turned out, they did, partly because of the incoming Delta variant of this virus, which obviously accelerated its transmission. In the second report we were tasked with the opportunity to look back as well as forward and think about the winter and beyond as to what steps might be taken to protect the community.

There were three things on which we particularly focused our attention. The first was the virus itself—we have had a lot of discussion about that today—and the possibility of variants of that virus emerging, and those variants accelerating the transmission and severity of the disease. That was one area. We had a section on the virus side, and you have heard a lot about that this morning.

The second was the impact that the virus would have, but also winter, on the national health service itself; that is the ability of the system to cope with more than a winter of which we have had experience in previous years. Part of the problem with that is the fact that, while we have been trying to boost immunity to this particular virus, there is a whole raft of other viruses that by the very actions we took, in terms of physical distancing, mask wearing, staying at home and all of that, never appeared last year. It is absolutely dramatic.

If you look at the graphs of all the other viruses—RSV, the respiratory syncytial virus, the influenza viruses and even the common cold viruses—they are all completely flat. Looking at those other viruses just last week, they are beginning to kick up a little bit. The difference that this has made has been huge, because obviously if you have a pre-existing condition, like asthma or chronic obstructive lung disease, and you get a common cold virus, for example, it causes a serious problem for those individuals. The emergence of viruses outside was the second area.

The final one is the ability of the workforce itself to be able to withstand another round of all of this. We have heard evidence today that, hopefully, it will not be as bad.

I work at Southampton General Hospital. We are seeing Covid cases in intensive care units at Southampton General Hospital. We need more evidence to say that these things are just picking up the virus incidentally, because certainly my respiratory colleagues are now being exercised by new Covid. It is nothing like what we had last year, but it is still there and that is an important concern.

We have a very distinct concern about the health of the workforce in terms of both the numbers, which we have heard a lot about in recent days, and the ability of that workforce to be able to deliver. By that, I do not mean just the hospital service but the primary care infrastructure as well.

Those were the three areas on which we concentrated. We made some recommendations that stemmed from those, many of which are being implemented. This is not in any way a critical thing, but the predictions we made about the magnitude of the hit, if I may use that term, in terms of the virus itself are probably less than we anticipated, but from the discussion you have just been having we will see where that pans out.

Q2581 **Chair:** Thank you very much, Sir Stephen, for that very good introduction.

Turning to Professor Chappell, you were good enough to sit through some of the previous evidence. You have heard that we have a comparatively high incidence of Covid in the population at the moment, but the difference between this winter and last winter is that vastly more people are now protected by the vaccine. Do you share Sir Andrew's concern that we ought to be driven by hospitalisation rather than the more superficial level of infection in the community?

Professor Chappell: I think we need to take all of those pieces of information into account when looking at where we are now and where we might be going, because they are giving us different bits of information. I think it would be a mistake to go for purely one or the other. We have seen a rise in cases particularly in the 10 to 19-year-old group; we are now seeing smaller rises in the parent group—the 30 to 49—and a small one in the older age groups. I agree that it is a balance between the number of cases and the wider impact, for example, on hospitalisations, the use of intensive care units for Covid and deaths, and I would suggest that we take all of those into account.

Q2582 **Chair:** But do you accept Sir Andrew's point that the rise in the prevalence or incidence of Covid is entirely predictable and that is what you would predict for the course of this virus?

Professor Chappell: I think it depends on where we are in the pandemic. We will see rises and falls as we have done throughout, but it is also very dependent on the intersection between vaccines, boosters, waning and behavioural changes. The modelling has been shared. We have modelling from SPI-M and the other groups. They all suggest that we are not heading towards a rise but that the numbers may be coming down. But it is modelling and is not a perfect forecast of what is going to happen. It is very much more about giving us some scenarios, but those scenarios depend on all the other human and population factors relating to vaccines and behaviour.

Q2583 **Chair:** Would you be worried about a continuing high level of transmission if the level of hospitalisation remained low and stable?

Professor Chappell: They are telling us different things and where we may need to input. For example, if hospitalisations remain fairly stable, we need to look at the impact of other respiratory viruses on hospitalisations and also on the indirect effects of Covid on the backlog. If we have an ongoing high number of cases, that will have some ongoing

forward transmission. That is why promoting both first vaccinations in older age groups and in the 12 to 15-year-old groups and booster vaccinations is so crucial, because vaccination reduces infection and if you do not have Covid you cannot transmit. They are all very interlinked, and the message needs to be clear that vaccination is one important part alongside the other components.

Q2584 **Chair:** Indeed, and that is the first recommendation in the advice of Sir Stephen's colleagues for winter preparedness. Nevertheless, we know that vaccines have an impact on transmission—on infection—and the severity of the disease. We have heard directly from Sir Andrew that protection against severe disease is very much greater than for transmission. Let us assume that we are going to go all out for vaccination. If the level of hospitalisation from Covid continues to be stable, would you consider a high level of infection in the community cause to call for or favour non-pharmaceutical interventions to be added?

Professor Chappell: I do not think we should be using infection in the community alone; it has to be considered with the whole package. What we are seeing now is consideration of all the information that we have from the surveillance. Dr Waite might want to talk more from that point of view on how best to inform the policy.

Q2585 **Chair:** I will come to Dr Waite in a second. It is perfectly easy, reasonable and obvious to think we should take all data into account; that is obvious. But decisions need to be made. If the level of protection against severe illness and hospitalisation continues, even though, as Sir Andrew expects, protection against transmission will fall over time, why should that even be considered a factor in contemplating non-pharmaceutical interventions?

Professor Chappell: I follow. I will draw a comparison. When we had no vaccinations, we knew that the level of cases was a good indicator of subsequent pressure on the health service. In this situation I can see that the level of cases does not translate into hospitalisation, but there are other factors such as the take-up of the booster. I am glad to see that 6.1 million booster shots have now been delivered and half of the over 50s. So continuation of that booster campaign against what Sir Andrew was describing in terms of waning immunity means we should not just abandon case counting as an indicator.

Q2586 **Chair:** You made a strong case that we should have the take-up of the boosters as being a significant factor, otherwise immune protection would wane, but if that continues or is positive what is the connection with case rates, if that is succeeding?

Professor Chappell: Seeing the increase in case rates gives us an indication some weeks ahead of where we might be going with hospitalisation. It informs the models that will then have us better prepared. We know from our colleagues, whether in intensive care units, emergency medicine or primary care, that we do not want to be acting in

the moment; we want to be as prepared as possible. As long as we are using that information in the round, rather than all of our policy being driven by the case rate only, which I do not see, I see it as being one component of the evidence that informs the policy making.

Q2587 **Chair:** To be clear, Sir Andrew, who has some eminence in these matters, having discovered the vaccine, expects his own vaccine over time to be less effective in preventing infection but to continue to be effective in preventing hospitalisation. If he is to be believed, it would be wrong to infer, would it not, that a rise in cases, which he predicts, should lead to alarm about hospitalisation?

Professor Chappell: Given that we are still within 12 months of the first vaccinations being rolled out, it is still early days to know exactly how vaccine waning and boosters will impact on hospitalisation. While there are ongoing discussions about how the testing programme continues, as Sir Andrew said, into next spring and beyond, for this winter it is clear that that information gathering on testing and the link to tracing and isolation of cases is still likely to be beneficial, given that it is not just vaccination; it is also mixing. While we see children mixing at the same rate as pre-pandemic, we have not seen a return to adult mixing. So there are more factors involved in transmission, infection and hospitalisation. Given that we are still 10 months into this new vaccination, I still think it is worth ensuring that we have a testing programme at this point.

Q2588 **Chair:** Do you agree with Sir Andrew's view that we should be moving away from testing asymptomatic individuals and testing instead people who are ill?

Professor Chappell: There is a short-term and a medium to long-term perspective to this. In the short term we should continue testing, particularly symptomatic individuals. I know that other groups are evaluating at what point we reconsider testing asymptomatic individuals beyond spring.

Q2589 **Chair:** The next decision is about when to turn off the testing of asymptomatic individuals, is it?

Professor Chappell: That is being considered, yes.

Q2590 **Chair:** It is a question of timing; it is when rather than if.

Professor Chappell: I would like to think that in five years' time we will not all be lateral flow testing.

Q2591 **Chair:** Five years?

Professor Chappell: Clearly, there is a stretchable point between now and five years.

Q2592 **Chair:** Sir Andrew referred to over the course of this winter.

Professor Chappell: This winter spans between now and January, and then beyond. I think that between now and January it is clear that we have committed to testing. We are then reconsidering where we go beyond January and beyond spring, but all of those need to react to the information we have available and consider where we go.

Q2593 **Chair:** Dr Waite, you heard what Sir Andrew said about people in hospital and those who have died within 28 days of a Covid-positive test. Many of them had Covid because there is a high level of infection in the community, but that was not why they died. Do you have some insight as deputy CMO into how that splits up in practice?

Dr Waite: I do not think I was in the room when those comments were made, but in terms of the overall burden of disease within hospital and the risk of coming into hospital or being diagnosed, yes, we can split that down into a number of different groups. That data is published by the NHS. First, there are those who are admitted with Covid at the point of admission. The NHS will also make available another analysis, which is the number of people who have been diagnosed with Covid while in hospital, but it is quite important to break that down further because being diagnosed the day after admission would probably mean you were Covid-positive when you came in and that underlies your reason for being in hospital, whereas being diagnosed, say, 14 days after admission would suggest you had acquired that infection while you were in hospital. Those are quite different groups.

Q2594 **Chair:** But the specific information required is whether someone who has died and tested positive for Covid within 28 days has died because of Covid or they have come in having had a heart attack or stroke.

Dr Waite: Sadly, the risk of death from Covid remains very strongly associated with age and increasingly with vaccine status these days. Having Covid on top of a number of other pre-existing conditions can be a precipitator for going into hospital.

Q2595 **Chair:** Sir Andrew made the point that it is important to have that data. If it all turns on the level of protection from hospitalisation, it is absolutely essential to know whether someone has died because they have had Covid, even if it is a contributory factor, or whether they just happened to have it and, sadly, would have died anyway. Given the huge importance of that, as deputy chief medical officer do you have access to that information and, if not, what are you doing to get it?

Dr Waite: We have some of the best mortality statistics in the world, thanks to the ONS and UK statistics. Those are not updated on a daily basis, as it takes quite some time to compile death certificates and look at the full details. Quite often, they will be cases that have gone to a coroner and so on and so forth, so there are good reasons why quite a lot of that data is delayed. As to the last ONS report I saw, I think they are updated quarterly, but I can certainly send you the most recent update as and when it is available.

Q2596 **Chair:** These are reports by the Office for National Statistics; they are not routinely reported by the NHS.

Dr Waite: The death statistics are compiled by the Office for National Statistics. We have two streams of data during the pandemic that we should not confuse. One is the very operational and very quick daily updates through the public-facing dashboard. That gives us a steer of where the trend is going. The much more detailed and fine analysis is the ONS quarterly updates, which we can rely on as being a kind of gold standard, but one gives us an indicator as to where the other might be going.

Q2597 **Chair:** Given how important this is, it is possible to gather data in other ways. You could have a sample inspection of records or interviews with clinicians to try to gauge this, could you not? If a lot is riding on how many people are in hospital because of Covid, surely given the importance of that and the fact we are spending over £30 billion on test and trace, it would be a sensible investment in information to have something bespoke to try to answer that question, would it not?

Dr Waite: I suggest this is not an indicator that will change very rapidly from day to day, so those ONS statistics would be sufficient. From the last version I saw, the majority were cases where people had died because they had Covid.

Q2598 **Chair:** Over 50% of people who have died in hospital within 28 days of a positive Covid test have died because of Covid.

Dr Waite: Certainly, on the basis of the last ONS update I saw. I can find the exact number. I apologise that I do not have it with me today, but it is still very relevant. The key thing is that the reduction in risk of dying from having a vaccine gets greater and greater in older age groups. If you are aged 70 to 79, being vaccinated reduces your risk of hospitalisation or death approximately to that of a 30-year-old. That is very important for understanding the pressure on the NHS going through the winter.

Chair: I will now turn to my colleagues.

Q2599 **Rebecca Long Bailey:** Thank you all for coming today to the Committee. Sir Stephen, you mentioned earlier the report of the Academy of Medical Sciences pointing to a number of other respiratory illnesses in the UK this winter. How worried should we be about this and the pressures that they will impose on the NHS?

Professor Holgate: So far, looking at the up-to-date data, we still have an extremely low level of these other viruses. This is two years now. First, what will obviously happen is that we will lose or have an accelerated decline in our immune response to all of these when they do eventually hit. Secondly, what we are most concerned about is influenza. Vaccinations for that are starting soon. The nature of that influenza virus when it does hit will be largely unknown. Normally, you have a clue because somewhere else in the world the influenza breaks out and your

vaccines are tweaked to be able to deal with that, and we can remember the different influenzas we have had.

We are in a bit of double jeopardy here. We have a waning population immunity to these other viruses and there is a question mark about the nature of the influenza when it hits, because it will eventually hit. I am afraid that at the moment we just cannot answer that. The chances that these other viruses will have an effect are very high indeed. For example, New Zealand, which is on the other side of the world, has been looking at RSV, for example. It has had a major resurgence there. We had an August-September resurgence here of RSV, though it is waning again now. Normally, RSV does not go up until the winter, so the next two or three months is going to be interesting.

Q2600 **Rebecca Long Bailey:** The academy's report suggested baseline interventions such as face masks and working from home where possible. Over the past week you have mentioned your own experience on the ground in hospital, but the British Medical Association is calling for those interventions now—plan B, if you like. What baseline interventions do you think the UK should be implementing now?

Professor Holgate: It is a really interesting question, because looking backwards—I will answer the question directly in a minute—we have come to accept that all of these other respiratory viruses like influenza should harvest so many thousand deaths every year. That is normal, and yet when we go to countries like Japan, Hong Kong or sometimes other south-east Asian countries people have worn masks, even outside the Covid era, so this is a cultural thing here. We have come to accept quite a high death rate. Two or three years ago we had a terrible death rate from influenza, which would have been protected against had people been wearing masks.

One answer to your question is whether this two-year experience has changed the threshold where people are prepared to do these things. For me, distancing in crowded places particularly, ventilation of rooms and the wearing of face masks are non-invasive things that people can do. We are trusting the community and industry, which you have talked about today, to do those things. Staying at home and working is another threshold. That is really where we might have some debate, because plan B has staying at home and working, plus these other things. I would myself put in first "plus these other things".

When people see others wearing masks they want to do it themselves. I came from Southampton today on the train. I was the only person in the carriage, which was three quarters full, wearing a mask. We cannot rely on the good will of the public to do this. We need some more positive direction. How that is shaped and progressed I am not able to say, but as we are going through this winter, with intergenerational mixing happening across the Christmas period, I would personally feel that. Our report says that. My experience as a respiratory physician is that we have not seen it yet and it will come.

Q2601 **Rebecca Long Bailey:** On the issue of the Government's plan B, so far the Committee has struggled to get clarity on what would trigger such a plan. Are you able to divulge any information as to what measures are being assessed at the moment, what the trigger points are and whether plan B interventions will be brought in in a phased way, just to provide a little bit more information? I think people watching from home today will simply want to know whether there will be a plan B and when it will be triggered. Can we start with Professor Chappell?

Professor Chappell: I will outline the overarching position and then hand over to Dr Waite for some of the specifics. It has been considered by SAGE and the SAGE minutes are available. It is clear, as we talked about earlier, that there is no single metric that would lead to it. There is a balance, which is why SAGE has provided advice and consideration of the possible measures and how they might mitigate in relation to each of the components of plan B. Because the impact of plan B is very much at a societal level, it is our view that we provide scientific advice and it is then a policy decision by Ministers. Dr Waite can talk more about the metrics themselves.

Dr Waite: I do not think that the metrics should be too much of a surprise to anybody. We have touched on case rates. Age-stratified case rates are very much more important than looking at just a single community measure. That is quite useful for a general understanding, but case rates among the 60-plus, 70-plus and 80-plus are much more likely to be associated with hospital admissions. But it is not just those rates and the number of people going into hospital on a given day; it is the speed of change of all of those. You could get to a given level of hospital admissions increasing by one or two a day over many weeks, or you could get there in a very rapid growth phase. That would cause quite a different assessment of the situation.

The third variable that tries to complicate those—I believe Professor Pollard was talking about this earlier—is that vaccination has changed those relationships, and waning immunity will further change them. For example, we might expect to see over time a greater proportion of people aged over 60, as they have a higher hospitalisation risk, going into hospital and getting infected if their immunity is waning. That will change again as the booster programme rolls out. An unknown at the moment is the extent to which that will further change the ratios between the metrics. Therefore, it is a total picture, but from my perspective the level at which society will say it will accept x number of cases or y number of hospitalisations is not a medical decision. That is very much a societal decision. That is why it is an important decision that Ministers and Parliament will take in England and the devolved Administrations as well, because it takes into account an awful lot of other factors. No interventions—certainly things like lockdown and so on—are without even health disbenefits as well as benefits, and there is also the wider economic piece about which we are certainly not the experts; we are

doctors, but we would feed in through SAGE, on which both Lucy and I sit, the advice to those who have to take the decisions.

Q2602 **Rebecca Long Bailey:** Is it the case that SAGE will be providing the Government with advice on when to trigger plan B interventions? A lot of the historical SAGE advice has not really given direction; it has just set out the picture. We are certainly exasperated within our communities as to what the trigger points will be. I understand that the science is being assessed, but I have not heard anything to date to suggest to me at all what the trigger points could be. Are they going to be determined by SAGE? Have they already been determined? Will they be published by the Government, as far as you are aware? Will there be any clarity at any point over the next few weeks?

Dr Waite: Lucy might want to say more about the role of SAGE in this, but this would be scientifically informed policy. SAGE will give advice and Ministers, absolutely, will have to make the decision, not us.

Another reason for that, as Professor Holgate touched on, is that other factors, even on health, would affect how you view the number of hospitalisations. If we see the extremely low levels of flu that we saw last year—there was almost no flu last year—that would rather change the impact on the NHS of, say, 1,000 hospital admissions a day, which I think we have reached relatively recently. Similarly, RSV has come down; it has plateaued. It will probably go back up. These are the two weeks of the year when we usually do see flu and RSV start to go up. There are a lot of other unknowns there. We can provide that advice, but we cannot say there is a single magic number for any of those thresholds because there is almost a web of interdependencies on all the other numbers. I do not know if you want to say more on SAGE, though, Lucy.

Professor Chappell: We should also focus on what we can be doing and should be doing now, not just focusing on a single trigger. As Tom says, it needs to be more complicated than that, because setting specific thresholds for x, y or z, if you did not consider them in the round, would not be appropriate. It would not be good science to say that it will be this number of this metric. I think it reflects the broader approach to assessing the situation. If it was as simple as a single metric with a single number that would be straightforward, but it clearly is not because of all the factors we have considered.

I think it comes back to what we can and should be doing now because plan B is what might be needed, but I think we are seeing everything going in the right direction in terms of take-up of vaccination, particularly the booster campaign but also going back to the first vaccination, which I know Professor Pollard and Professor Holgate have described as being key, and the communications around that both for older age groups and younger.

I share the view that we should all be wearing face coverings where we can—on trains, tubes and crowded spaces—where it makes sense and is

already part of the recommendations. As all of us have described, the next step up that has impacts much more widely on the economy needs to be taken with science advice informing policy rather than on a single number.

Q2603 **Rebecca Long Bailey:** That is very helpful. SPI-M, as I am sure you are aware, stated in September that, "If enacted early enough, a relatively light set of measures could be sufficient to curb sustained growth. During a period of sustained epidemic growth, however, the more stringent the measures introduced, the shorter the duration needed for the measures to be in place to reduce to a given prevalence."

Do you think there is a risk that if we do not enact parts of plan B now we are looking at more sustained periods of more stringent measures in the future? I put the same question to everyone on the panel.

Professor Holgate: I do, yes. I cannot speak on behalf of the academy now because all of this was not available then, but speaking as a respiratory physician on the frontline in Southampton the answer to your question is, yes, I do.

Professor Chappell: I think it suggests that plan A, plan B and whatever plan C looks like are mutually exclusive, but they are not. There is overlapping, for example, in the use of face coverings. We do not have to wait for a trigger. I completely accept Professor Holgate's point about the communication of it and asking individuals to think about the risk and how they can protect themselves and others. We do not have to wait for a trigger to enact sensible behaviours that will reduce it, so it is a question of how we ensure that that message is communicated. It is a real strength that we now have much more transparent data that people can follow, but there may be a point at which Ministers do make that decision, and that is being constantly assessed by both SAGE and Ministers.

Dr Waite: SAGE has been very consistent in its advice that the earlier you act, the greater the impact of any intervention. At the same time as the September statement, SPI-M undertook a very useful review of what had happened over the summer, the disease trends. We are very much at the lower end of some of its July models in terms of the number of cases. What we learned from the summer is that it takes fairly small shocks or nudges either up or down to push R and thus the growth rate above or below one. You are either in a growth phase or a shrinking phase. That is quite reassuring in a sense, because fairly small steps that we can all take—making sure you meet outside where you can, or in a well-ventilated place, wearing a mask, keeping your distance, following good hygiene and so on—will help push things into either a slow downward phase or mitigate a slow upward phase. Of course, the point is that that would be a fairly slow downward pressure rather than some of the very sudden drops we have seen around the larger and more impactful but very costly interventions over the past 12 months.

Q2604 **Chair:** Professor Chappell, is there a plan C?

Professor Chappell: It has been proposed. The name has been mentioned. It is not being extensively worked up. Tom may know more from the UK Health Security Agency. People have used that phrase.

Q2605 **Chair:** In the Department of Health is there a set of measures beyond plan B that is being put together?

Professor Chappell: At the moment the focus is on plan B.

Q2606 **Chair:** Is there one being prepared beyond plan B?

Dr Waite: I have not been consulted on anything about a plan C.

Chair: You do not know anything about that, Professor Chappell.

Q2607 **Katherine Fletcher:** I want to talk about variants. Basically, we need public permission at this stage to engage in anything. It strikes me that we need to give the public information and data to gain that permission, because for the past 18 months it has been having a massive impact on lives and livelihoods. The British public have been marvellous. We have heard evidence on this Committee about how they have engaged in literally life-changing measures for months when the expectation was not that that would be the case, but I sense there is a subset of people who, quite frankly, have had enough of doing that. It was probably visible on your train on the way in. I had Covid for the second time; I have been double-jabbed. It is really unpleasant, so I will stick a mask on, but I can get tutted at for virtue signalling about using a mask. So I am going to be deliberately challenging about the things that might give the British public the information they need to change those thoughts and views. One of them is actual data.

If I may pick up the thread with which the Chair started, normal people will say that everybody in intensive care has not been vaccinated; the vaccine is available to them and there is an element of personal responsibility for that. To be clear, that is not my view, but it is one that has been expressed to me. This gap between people dying of Covid and people dying with Covid is being used as a slight justification by some people.

Dr Waite, there has to be a middle ground between three-monthly ONS data and the kind of quick and dirty data that gets put together by the NHS to give us an emerging trend. I know you have answered the Chair, but can we just return to that? If we are to start asking people to do something they are resistant to, the first thing we will need to do is say why and in numbers.

Dr Waite: I agree. I may be wrong; the ONS may give a monthly update. I certainly know that it is a routine publication. I think the middle ground that you might be talking about is not necessarily going to the absolute extreme end, which is mortality from coronavirus, but, as you say, the admissions to ICU and hospital can be very life changing for

people, particularly those who may already be unwell. Even in your 20s or 30s, being admitted to hospital and to intensive care can be a long, slow road to recovery after that. Those figures certainly are put together every day and published. The important way of getting to the hearts and minds piece that you were talking about is trying to explain to any given age group the reduction in their risk from being vaccinated, because it is not the same as you go through the age groups.

Q2608 **Katherine Fletcher:** Forgive me, but we are talking about the almost immediate imposition of a set of measures that some people find unpleasant—for example, face masks on trains. It is not the debate about vaccination; it is a debate about how we take the British public with you on recommended advice. Vaccination is important but separate.

Dr Waite: The raw numbers for those admitted to hospital, for example, are published every week by PHE. I believe they are published in what is called a vaccine surveillance report, but it gives you in a given age group—I have it here somewhere—the number of hospital admissions in an age group and, of those, how many are double-vaccinated and how many are unvaccinated, and you can make comparisons between them.

Q2609 **Katherine Fletcher:** But it is not dying of Covid versus dying with Covid or the long-term effects of Covid that I am aware of.

Dr Waite: To drill down a little further on "dying with Covid", quite often having Covid will be the precipitant for your heart attack or super-added pneumonia; it is a significant contributor. I do not know whether Lucy wants to say more about those. Yes, it is a part of your admission, but that is no different from many other infectious diseases. Take flu as an example. A large number of people who come into hospital in any year, apart from last year, may have flu as one respiratory infection, but a pneumonia on top of that can unfortunately be the final straw, if you take that analogy.

Q2610 **Katherine Fletcher:** I completely accept that, but in a normal flu year we are not asking people to do stuff they do not want to do if they perceive it to be injurious to their lives and livelihoods, be it changing the way their businesses operate or changing their personal habits. I do not understand why we cannot create a publicly accessible dataset that allows for this interrogation and maybe some myth busting.

Professor Chappell: I do not think it is a binary death from Covid versus others, because there is a leading cause of death on a death certificate and there are contributory factors. For example, I have the September stats here. Dementia is the leading cause of death; ischemic heart disease is the second cause of death; Covid is the third. There are other similar levels for lung cancer, cerebrovascular diseases and chronic lower respiratory disease, but the extent to which Covid is contributory will be varied across all of those for all the reasons we have heard. I share your views on publishing data where possible, but sometimes it is easy to present it as all or nothing when it is not. For those who are in

hospital—all of us have experience of this as doctors—it does matter at one level, but, if your relative has died, the exact leading cause is probably less important than the fact that they have died at this time. That is what they say to me.

Q2611 **Katherine Fletcher:** I personally am not arguing with you. I am trying to explore a mechanism by which we can have a better debate. Sir Stephen has just given very clear views as to where we need to go back to stuff that a lot of people are resistant to and we have to find a different way of talking to them. The uncertainties within data are one thing. I am a little bit away from my brief. I appreciate you asking, but maybe it is something to which you can give further consideration.

I want to come back to the other thing that will be a very strong and powerful trigger for people to do NPIs again, which is either a differential threat—the emergence of new diseases or an increased virulence of existing diseases due to waning immunity over lockdown—or new variants. If we come back to new variants, we have heard from Dame Sarah Gilbert that she believes it is unlikely we will have a full vaccine escape variant. Can I confirm whether that view still holds in your mind, Sir Stephen?

Professor Holgate: I am not a virologist. I will say that to start. We have the AY4.2 around now, which incidentally and interestingly came out of India, as Delta did, so it is just a branch of that Delta virus. It is said to be 10% or 15% more transmissible, but the vaccine is effective against it.

We have been concentrating very much on the spike protein on the virus, which is its way of getting into cells. What this virus has done is learn how to avoid what we call the innate immune response, which we have not talked about at all. We keep talking about the adaptive immune response, which is all about vaccines. The innate immune response is why children are not getting the disease, and why older people, obese people and people with diabetes are getting the disease, because they do not have that initial ability to eliminate the virus when it first comes in.

It comes back again to fomites versus aerosol. The reason why the aerosol is so damaging is that it gets to the parts of the lung where fomites would not get to. It penetrates and that is why it is more severe.

The important thing here is to think about variation not just in terms of a single group of proteins that everybody is focused on at the moment, which is the way the virus gets into the cell, but all the other proteins that can confer upon a virus an ability to get round the immune response. So far, there have not been any in those other multiple proteins, but that does not mean there will not be. That is really the point. We can go only so far in predicting from what has happened in the past and trying to learn from other viruses, but I would not be at all surprised if somewhere in the world another variant turns up.

Q2612 **Katherine Fletcher:** Do you think we have done so well with vaccines that we are creating a massive selection pressure?

Professor Holgate: Yes, exactly. That was the next thing I was about to say. By generating these highly effective vaccines, which is super—we keep talking about them—we have put selection pressure on a Darwinian system. Although viruses are not really truly living things in the way we think about life, they are mechanically able to mutate randomly until they get round an obstruction.

Q2613 **Katherine Fletcher:** It is a volume game, and they have the volume.

Professor Holgate: That is correct. I am not saying it will happen, but to suggest it will not happen is quite a bold thing to say.

Q2614 **Chair:** That is a fascinating and important point. On this point, Dame Sarah Gilbert takes a more optimistic view. She says that, if the virus changes its spike protein so much that it cannot interact with the ACE2 receptor on the surface of a human cell, it will not be able to get inside the cell, which leads her to conclude that she does not think there is an enormous amount of concern that we will see a switch to something that evades existing immunity.

Professor Holgate: Yes, and that is all about the spike protein, but what about the mutations in the other 75 proteins that are not the spike protein, which confer the ability of this virus to evade the human immune response?

Q2615 **Chair:** So you are more worried than Dame Sarah.

Professor Holgate: The spike protein is fine, and everyone has concentrated on that as if that is the only way the virus is able to mutate. What this virus has done in the bats and other animals it has been in is develop mutations and changes that have enabled it to live in those animals like fruit bats without causing disease, but it is only when it gets out of those particular animals and into the human that our immune response is not able to cope with it. If this virus starts to learn some tricks to get round the vaccine and the other immune responses that we might be able to generate, there could be issues. It is quite difficult to know in this random mutation world, which is what is happening here, how a pressure selection might confer an advantage on the virus to get round it. I am saying that only because I am talking now as a respiratory physician, not a vaccinologist.

Chair: That is why it is a particular privilege to have you here with both hats on.

Q2616 **Katherine Fletcher:** It is very easy to enter into the science of it. I am continually wheeling myself back to the fact it could happen, but people do not want to wear masks on trains because of a "could".

Professor Holgate: They do not.

Q2617 **Katherine Fletcher:** That is the basic trade-off that we have. Are you modelling for potential new variants this winter and the need for a completely novel vaccine?

Professor Chappell: There are two aspects to it. One is the modelling, which can make assumptions with and without a variant. The other is what infrastructure we have that will help us. Some of the infrastructure was here before the pandemic. The UK vaccines network allowed us to be well ahead of the game in stepping up, as you have heard, with the Oxford-AstraZeneca vaccine.

The other component of this is the genomic sequencing that is going on and the extensive networking through the WHO and others to understand what is going on with variants, and the very intensive daily work that is done on assessing the risk of those variants, which is why they may move to a variant under investigation or a variant of concern.

There will be a range of opinions. They may all be right or wrong, but what matters is: are we watching out for it? I think that the surveillance we have in place through the UK Health Security Agency, other mechanisms and the more international look means that we are certainly better placed than we have been. I guess there is a real move within Government to think about the 100-day mission and the bigger pandemic preparedness through the rest of this pandemic but also in preparation for others.

Q2618 **Katherine Fletcher:** Fair enough. Dr Waite, would you draw that one in?

Dr Waite: I will try to step out a little bit into population health rather than the virology of it, but there are three key aspects of variants. There is almost a triangle of things that can alter their effect or impact on the population. You mentioned one, which is whether or not a new variant escapes the vaccine. The second is whether or not it is more transmissible. The third is whether it causes greater or less disease than other variants.

We have seen some variants already emerge that have had a degree of vaccine escape. I think everybody will remember Beta emerging quite some months ago now. However, if it cannot compete with more transmissible variants that are already in circulation, Delta being the big one at the moment, it just has sharper elbows; it pushes the more vaccine-escaping but less competitive variant off the pitch. That is important to think about in terms of the global burden of the disease. Delta has pushed a lot of things out of the way at the moment because it is more transmissible. Those three aspects affect one another.

The other point on vaccine pressure is that, historically, we have managed to reduce the burden of a great number of diseases through very successful vaccination programmes. I do not see any reason to think that this would be different.

Q2619 **Katherine Fletcher:** That is a very fair point. To summarise it—please

tell me if this is not the case—if you have not had a jab, get it because you are really at risk, but, if you can, please start to think about reducing transmission in the run-up to winter while we are assessing some of the other things that are going on.

Dr Waite: To add to that, many of the people we vaccinated at my clinic on Saturday were having their flu jab and their Covid jab at the same time. That is really important for this winter.

Katherine Fletcher: That is a really good point.

Q2620 **Chair:** To reflect on where we are, looking into the winter, most years the NHS faces winter pressures. Running the NHS during the winter is never easy. The figures for the middle of October for the people in hospital with Covid were just over 7,000, which is about 11 per parliamentary constituency. As we have established, some of those will have Covid, but that is not the reason why they are in hospital.

Is there a danger going into the winter with the pressures on the NHS that having uniquely the tools available and the Coronavirus Act to be able to impose restrictions on people's behaviour—measures such as advising people to work from home—we reach for them because they are available, when the contribution that Covid might be making this winter is significant but no more than, say, flu in a bad flu year would make, or some other disease in another year? Are we being rigorous in not reaching for measures that may be helpful but we would not have introduced this winter were it not for having done so before?

Professor Chappell: I think you describe the tension between considering the Covid-only piece and not just the direct but indirect effects both at an individual level and a population level. There is a considerable amount of work across the Department of Health and Social Care to consider the wider impacts and not to see it just as an isolated problem. That will range from the work being done to tackle the backlog; to look at elective recovery; to look at work on the diagnostic strategy to increase availability through the diagnostic hubs and other routes; to look at how we work towards earlier cancer diagnosis; and look at where the impact is greatest with the indirect effects of Covid, and so where we want most catch-up, for example in diagnosis of hypertension.

You are right that at this stage we are very much moving to a place where we are trying not to consider it just on its own. We have additional measures that are both available and may or may not be enacted as policy, but across the Department of Health and Social Care there is a very clear view that it needs to be considered with the direct and indirect effects as a whole, not in isolation.

Q2621 **Chair:** In a previous session, I think last autumn, we discussed the impact of the Government's scientific advisers warning the Government that the NHS would be overrun. That was very powerful. It was very difficult practically to gainsay that. Is there a sense within the Department of Health—perhaps Professor Chappell as chief scientific

adviser and Dr Waite as deputy CMO can answer this—of the degree of responsibility that needs to be exercised in making an argument that the NHS will be overstretched, because that is the use of a card that is very difficult to gainsay? Is there a sense of just how important that is?

Professor Chappell: I think it is a strength of the approach by Sir Patrick Vallance and others that scientific advice is now more tapped into than ever before through the network of chief scientific advisers and others, and calling on multiple groups through SAGE and many other committees, whether it is NERVTAG or JCVI. We have really seen that come to the fore. We have seen clearer use of scientific advice and more transparency about those decisions than ever before. We should welcome that input.

The transparency with which SAGE provides advice and publishes it is clear, but we should recognise that the scientific advice is one component and that the societal response and the responsibility of elected politicians to make those final policy decisions is also clear. There is space for both and we continue to do that now as before.

Dr Waite: In the conversations that I have there is the greatest awareness of what we mean by NHS pressure; that is, people who are waiting to be admitted to A&E and those who are going into hospital because they are ill for any number of reasons. That is very much at the forefront of an enormous number of conversations and is one of the reasons why monitoring not just coronavirus admissions but the other respiratory infections this winter—all of the non-communicable disease admissions to hospitals, emergency and elective—is important. Therefore, it is not just looking at a single metric, say hospital admissions, but thinking about the entire pressure on the system—people waiting for ambulances, people waiting for discharge and so on—and seeing the NHS as a sign that people are unwell and require hospital treatment, rather than seeing it as a service to be protected. It is a service that is there and is busy because people are unwell, and that is very important to remember.

Chair: Perhaps I can thank Sir Stephen, Professor Chappell and Dr Waite for giving us the benefit of their expertise and practice. We are very grateful for their evidence to the Committee today. That concludes this session of the Committee.