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Transkript

„Eigenschaften und Verbreitung der SARS-CoV-2-Variante B.1.617.2“

Experten auf dem Podium

Prof. Dr. Ravindra Gupta
Professor für klinische Mikrobiologie, University of Cambridge, Vereinigtes Königreich

Prof. Dr. Neil Ferguson
Direktor des MRC Centre for Global Infectious Disease Analysis, Imperial College London, Vereinigtes Königreich

Dr. Annegret Burkert
Redakteurin für Medizin und Lebenswissenschaften, Science Media Center Germany, und Moderatorin dieser Veranstaltung

Video-Mitschnitt

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https://www.sciencemediacenter.de/alle-angebote/press-briefing/details/news/eigenschaften-und-verbreitung-der-sars-cov-2-variante-b16172/

Transkript

Moderatorin: *[00:00:00]*
So, we would like to start with today's press briefing of the Science Media Center Germany. My name is Annegret Burkert. I'm an editor for Medicine and Life Science here at the Science Media Center Germany. And the topic of today's press briefing is the SARS-CoV-2 variant B.1.617. This variant originally evolved in India. And we all know these dramatic pictures that we have seen during the last weeks where the second wave of COVID-19 infections has led to thousands of new infections every day and several severe disease outcomes. And during the last weeks, the variant has also spread to other countries and can be found now in many places all over Europe, and especially in the United Kingdom the arrival and the spread of this variant B.1.617 has been tightly monitored and the virus has been characterized. The two experts here today are involved in this monitoring process and in the analysis of B.1.617. This is Professor Neil Ferguson, he is the director of the MRC Centre for Global Infectious Disease Analysis at the Imperial College in London, and professor Ravindra Gupta who is a professor of clinical microbiology at the University of Cambridge. I'm very happy that you are here with us today and that you are willing to share your insights on this new virus variant. And before I start, I would like to point out that you attendees can now already start to ask your questions in our question-and-answer tool. For our British colleagues who joined today, I would like to point out that we don't take your questions directly, but please put your questions in this question-and-answer tool and then my colleague will forward them to me. And to our German colleagues, we would be very grateful if you could ask your questions directly in English, that would make it much easier for us. So, I would like to start with the question to Professor Ferguson, who is and has been involved in the modelling of this SARS-CoV-2 distribution in Great Britain. How do you currently evaluate the growth of B.1.617.2 In the U.K. during the last weeks? And how is the current situation? Are you concerned?

Neil Ferguson: *[00:02:20]*
So, I mean, like B.1.1.7, the Kent variant, before it, what we're seeing now is, this Indian variant B.1.617.2 quite rapidly replacing previous variants and circulation like the Kent variant. And that growth has been fairly consistent in the last four weeks or so. I think there's a big difference, though, comparing the situation we're in now with the situation which held back in December of last year when we were looking at the Kent variant replacing. They make infection that will go very, very low at the current time. And so whilst we're seeing this variant grow quite quickly, it's very hard to estimate really how much of a transmission advantage it has over the Kent variant, because effectively they're circulating in quite different population groups. The Kent variant is quite still generally distributed across the population but at very low levels. Whilst this new Indian variant came in through imports into a small subset of communities across the country where it's growing quickly, then. So that sort of situation makes it difficult to be definitive about how much more transmissible this variant is compared with what we've seen before. What we can say, though, it is definitely more transmissible, and it could be anywhere from 20 percent to 80 percent more transmissible. But we really can't pin a number on that at the moment.

Moderatorin: *[00:03:52]*
So the Kent variant, because I think this term is usually not used here in Germany, is the B.1.1.7 variant, right?

Neil Ferguson: *[00:04:00]*
B.1.1.7, yes. So, B.1.1.7 was considerably more, probably 60, 70 percent more transmissible than variants which came before it. We think this Indian variant B.1.617.2 is more transmissible than B.1.1.7, but we can't exactly quantify how much at the current time.

Moderatorin: *[00:04:25]*
And to my last question, are you concerned? How do you evaluate this growth that you see during the last days or weeks?

Neil Ferguson *[00:04:35]*
I mean, we'd be in an easier position if this variant had not arisen, undoubtedly. And so, I mean, there are some concerns. I think we're in a much better position, I'm talking about the U.K. now, than we were five, six months ago when B.1.1.7 arose. First of all, we're starting from much lower infection levels but also, we have vaccinated over half the population. And so, vaccination will make a substantial difference, even against mutated strains like the Indian variant. Maybe we'll come on to that later. But we think, whilst there probably is some effect of the Indian variant on vaccine efficacy, still vaccine effectiveness against particularly against severe disease is still going to be very high.

Moderatorin: *[00:05:33]*
There is a follow-up question directly: Is there a slight increase in the still low incidences in U.K., and can this slight increase now be related to B.1.617.2?

Neil Ferguson: *[00:05:49]*
Yes, I mean, it's the simple answer to that. There is this slight uptick now in infection rates in the U.K. and that is down to B.1.617.2.

Moderatorin: *[00:06:01]*
All right, Mr. Gupta, you have analyzed the variant already in the lab. How does it discriminate from the Kent, the B.1.1.7 variant?

Ravindra Gupta: *[00:06:13]*
So the B.1.617 variant has a couple of different sorts of types, one is a B.1.617.1 and there's another one, B.1.617.2, and then there's a third. But the two main ones are different in a few ways. But there's one key change in the receptor binding domain, which is on the surface of the virus. And by comparing the two, the B.1.617.1 and the B.1.617.2, they look very similar, but for some reason the B.1.617.2 seems to have dominated infections here in the U.K. and in India. So, there must be something special about the B.1.617.2. Looking at the way that neutralizing antibodies that are contained in blood from vaccinated individuals, how well that is able to deal with these artificial viruses that represent the B.1.617.2 we can see that there is a moderate reduction in susceptibility of the virus to those antibodies. So, there is a difference. It's a little bit less sensitive or more resistant than the B.1.1.7. It's not quite as resistant to

antibodies as the South African variant B.1.351. So, somewhere in the middle, somewhere sitting where the Brazil variant, for example, sits. So, it does have the ability to make antibodies less effective. And that's important because, I think, the reason that we're seeing growth potentially is that the virus not only can do some partial evasion, and that's important because so many people are vaccinated now. And in India, there was a quite a high proportion of people who had previous immunity because they've been infected in previous waves. And then there is a mutation in that virus that is not about evasion. It's about increasing the infectivity of the virus. And we're still doing some work to show that we think that the Indian variant has a mutation that makes it much more able to infect cells and to transmit between cells quicker. So, in other words, that might lead to greater viral load, might lead to greater transmissibility.

Moderatorin: *[00:08:08]*
So do you think that this variant has an advantage in the vaccinated or immunized population versus B.1.1.7, and would the picture be different, like, for example, in Germany not so many people have got two shots yet, the full vaccination. So, is it likely that, for example, this variant would not be as dominant in a country like Germany where not so many people are immunized yet?

Ravindra Gupta: *[00:08:34]*
Yeah, that's a really important point, that is the relative advantage to do with the immune escape or immune sort of evasion. I think, that's potentially part of the explanation, although we're seeing theB.1.617.2 Indian variants in unvaccinated individuals as well. And the question about the first dose, you know, yes, there are data the PHE, Public Health England, released showing that a single dose is not that effective at stopping infection. And that level of infection is reduced for the B.1.617.2 variant and even further. So, that fits with the in vitro data. After the second dose, the levels of antibodies are very, very high. So, there's really good protection across the board, again, there is a small reduction for theB.1.617.2 variant. So, yes, in somewhere like Germany or in populations where there's partial immunity, either from previous infection or low levels of antibody, then the virus will have that nice sort of in the sweet spot of an advantage of immune evasion plus greater transmission. You've got to remember that some of these mechanisms for increasing infectivity of the virus will also help it to escape antibodies as well. I won't explain it now, but it's to do with whether the virus moves from cell to cell without having to leave the cell and therefore the antibodies can't reach those viruses. So, yes, I think that the virus will have an advantage in under-immunized or partially vaccinated individuals.

Neil Ferguson: *[00:10:04]*
I want just to add to that a little. So, undoubtedly a degree of immune escape, and we can't quantify precisely what effect that's having on transmission that will help the virus in a country such as the U.K. But if we look at data from India and actually looking at the U.K. data, it can't be the whole picture. It's almost certain that this virus has an intrinsic transmission advantage on top. I mean, it's more transmissible even in people who haven't been immunized, who

haven't had a prior infection. But we can't say whether that's the kind of 10 percent increase or a 50 percent increase at the current time.

Moderatorin: *[00:10:45]*
So, in your last preprint, Mr. Gupta, you also mentioned a case from an Indian hospital where health care workers who were fully vaccinated had an infection or infection group with this B.1.617 variant. So if fully vaccinated people start to spread the virus again and the virus again is more transmissible and then also can reach unvaccinated people, what does this mean now with all your strategies of opening up? How do you evaluate this whole situation?

Ravindra Gupta: *[00:11:23]*
Yes. I mean, seeing the cluster we described, I mean, I was very surprised by it because that's a single virus that is transmitted to twelve people with very few changes in suggesting it's a single transmission event. So they were potentially in the same room as each other. They were not wearing masks, and the transmission happened despite full vaccination, and there was enough virus in those individuals to sequence, which means it was a decent amount of virus. Fortunately, nobody got very sick, so the vaccines are still doing their job. They're still protecting you from severe disease. But as you said, the worry here is that this is an avenue for the virus to persist in a population and then to reach unvaccinated people or vulnerable people in society. And in the context of opening up … I believe that we should potentially be, let's say, allowing vaccines to have the full effect, which means waiting a little bit longer to reach more people, get second doses into more people so that the virus has a very, very strong barrier against it. Right now, we have an issue where we are increasing social contacts in the face of an expanding growth of the virus. So, that gives the virus an opportunity to really seed itself within the U.K. population. And it's going to be very difficult to get rid of it once it's there. That's my opinion.

Moderatorin: *[00:12:51]*
The next question is what, if anything, can the data from India itself tell us about transmissibility and vaccine escape? Are you mentioning the genomic surveillance data from other countries in Europe, such as Germany, where the B.1.617 variant makes up about two percent of new cases? This question, I got in, and I don't know if you got it correctly. I didn't get it [straight away].

Ravindra Gupta: *[00:13:21]*
Well, I think that what they're saying is that it looks like that the Indian variant is at low prevalence in European countries. But, yes, that may be just because there have been a few importations. It might be that because of vaccination coverage is lower than the competition between B.1.1.7 and B.1.617 is maybe a little bit more even. It's probably different populations, maybe the surveillance of course, the surveillance in Germany is – I cannot remember what proportion of infection they are sequencing, but it may not be as high as some other areas. So, there are lots of reasons. I wouldn't necessarily be reassured by the fact that it's low in country X or country Y at the moment. It's all about what's happening, what the restrictions are, what the vaccine coverage is. You know, those things will feed into the long-term outlook. And I think learning from India is important, although we don't have very good

serology data from extensive areas to know what the background immunity level was, or the previous exposure was. Obviously, vaccination is very low in India. So, the findings in India could very well be explained by transmission in unvaccinated people who have not been infected before. But I think there is an element from what we're hearing from reports from various people, family members, that people who have had previous infection are getting infected again, many of them are getting severe disease, and that includes people who have been partially or fully vaccinated as well. So, yeah. We should take notes, that we should take it seriously, is what I'm saying.

Moderatorin: *[00:14:57]*
Yeah, Mr. Ferguson, are you also monitoring the genomic surveillance data from other European countries, for example, such as from Germany and include them in your modelling, and what can you see there currently?

Neil Ferguson: *[00:15:13]*
So, mostly we're not so much modelling the data, but just analyzing it in conditions, statistical methods. I mean, as a team, we're looking at data from across the world. I would say, … you can't say much from most of the European countries apart from the U.K. because the numbers of sequences are so low and the first recorded case is so recent. We have no data with which to really assess the trajectory of how quickly it is expanding. And, as my colleague just said, I mean, there are delays in the system in surveillance, and surveillance varies from country to country. So, we will have to almost certainly, just as we did before with B.1.1.7, we will have to wait a few weeks to really see what trends we see across the continent of Europe. The U.K., because of its historically high levels of connectivity with mobility, travel to and from India has been much more heavily seeded than any other country in the continent.

Moderatorin: *[00:16:19]*
There's another question to you, Mr. Gupta, and I will directly turn it to you, since you need to leave a bit earlier. It is interesting that the B.1.617.2 variant, which seems to be more transmissible, lost one mutation. It's a E448Q mutation which was before feared to be involved in the immune escape. Do you think there is something about immune escape mutation somehow hinders transmissibility?

Ravindra Gupta: *[00:16:47]*
OK, yes. In theB.1.617.1 variant which is more closely related to the initial ancestor of the viruses, there was this E484Q mutation, which is, you know, in the same place that a E484K was. And that's the mutation found in the South African, Brazilian variants, respectively. So, we know that that's a mutation we don't want to see. The fear was that this would add onto the L452R mutation and make this sort of double mutant, and that's why there was all this double mutant sort of panic in India for a while. We showed in vitro, actually, that the effects don't add up together to make it a sort of really super resistant mutant, you just get this sort of same level. And that may be why the E484Q has been lost in theB.1.617.2 because, actually, from the data we've generated, the E484Q does not seem to add any elements of additional immune escape onto the existing virus. So maybe it had some deficiency or maybe it was lost randomly and whatever. It seems that T478K was acquired instead of it. And when we've

tested T478K, it also doesn't seem to add, you know, a huge amount onto that immune evasion aspect, but it may have other roles. It may have tied to ACE2 binding. You know, there are other aspects of its biology that we haven't uncovered yet. So E484Q probably wasn't that beneficial for the virus.

Moderatorin: *[00:18:17]*
Mr. Ferguson, you mentioned travel restrictions before, so you have mentioned that the Indian variant came to the U.K. through travel, and some European countries have now imposed restrictions on travel from the U.K. now. From an epidemiological perspective, do you think such travel restrictions are good measures to limit the spread to continental Europe? And if so, should all European countries take the step?

Neil Ferguson: *[00:18:44]*
I'm not sure if I'm going to directly answer that question. I mean, travel restrictions. It depends on what they do. I mean, they at best, they're likely to slow spread unless they're very draconian. They're not going to be successful at keeping these things out for ever. And that's the history we can see across the pandemic. So, yeah, the Indian strain is now quite widely distributed across the world in dozens of countries, and people's travel patterns involve going from one country to another. And so, yeah, some restrictions on travel in the U.K. may slightly slow the import of cases into European countries. But I doubt that it's going to have a very large effect, partly because infection levels at the moment are still very low in the U.K.

Moderatorin: *[00:19:39]*
Another point that I am very interested in, so there seems to be an enhanced spread in schoolchildren in India, and there were also the warnings from Singapore that the B.1.617 variant is more pathogenic in children. Can you confirm those signals?

Neil Ferguson: *[00:19:57]*
I can talk to the epidemiology. So, there's a hint in the data that the under twenty-ones are slightly more likely to be infected with this variant compared with other variants in recent weeks in the U.K. Whether that reflects a change in the biology or reflects what's called founder effects or the context that people who came into the country with the virus made a seeding infection in certain schools, colleges, that's impossible to resolve at the moment. We have a signal, but we can't really interpret its meaning in terms of biological hypotheses. As to pathogenicity, as to how severe infection is, we really have no data on that, and there's no signal that I'm aware of that this is more severe in children than other variants.

Moderatorin: *[00:20:56]*
Mr. Gupta, do you want to add something to if B.1.617 is more pathogenic in children?

Ravindra Gupta: *[00:21:04]*
I think, as my colleague Neil said, it's difficult to establish at the moment, it's too early, I think. But again, I do think we should take report seriously because that's, you know, the first sign that you have of a problem. And often, if you wait too long for the right data, it's too late. So,

we should be receptive to what's coming out, hopefully the countries where they're seeing this will be studying it in a kind of rigorous way so that we can get that information. I think, if it were true, I think, we may have seen it from India already. So that's what, sort of, makes it more complex. But then again, I think maybe many schools have been uncomfortable with the sort of closure system has been for schools, but many states have had lockdowns and cities have had lockdowns which include school closures. So maybe the opportunity was to see it.

Moderatorin: *[00:21:55]*
There's another specific question to you, Mr. Gupta, from a journalist who is saying: "I just talked to a Swiss expert, and he said that while the B.1.617.1 variant has immune escape the B.1.617.2 variant hasn't, referring mostly to sequences. Is this correct so far?"

Ravindra Gupta: *[00:22:17]*
... So, when we make the viruses in vitro they seem to have similar effects, one may be milder than the other, but sometimes it's difficult to tell because you're testing these viruses against serum or antibodies from different donors and that introduces some variability. And different people may get different results because they are taking antibodies from different populations. You know, we all make different antibodies. You can't really look at the sequence and say, that one is more resistant [than] the other, because we don't know much about those key mutations. We know very little about T478K. We know very little about 44Q. And we don't know much about the way they are combined with L452R, the other key mutation. So, I would suggest that we are at an early stage of our understanding, and we need to do more work.

Moderatorin: *[00:23:06]*
All right, Mr. Ferguson, is it possible that B.1.617 has so far spread more in environments that favor greater spread? So, we know that the spread in the U.K. is not really equally, and that the measures' effect is less on variant characteristics in these areas?

Neil Ferguson: *[00:23:28]*
Yes, very good question. And that's what I was trying to point out, because it has been seeded into certain communities in the U.K. in some cases with quite large multigenerational households, quite high population density. Some of the explosive spread we've seen in recent weeks may be down to the fact that the community groups it's been seeded into just have higher contact rates. In the coming weeks, as we see this variant spread into the wider population, we should be able to resolve those issues. But it's still going to take two or three weeks more time before we can get a complete picture.

Moderatorin: *[00:24:11]*
Do you know if those groups, have they already been vaccinated or are they largely unvaccinated?

Neil Ferguson: *[00:24:20]*
So, I mean, there's some variation in vaccination levels across the U.K., but still we're talking about populations with overall quite a high level of vaccine coverage. We're talking about

differences of maybe five percent in coverage between different areas, not enormous differences. So that doesn't explain the rate of growth we've seen.

Moderatorin: *[00:24:44]*
And [like] what [kind of] measures should be taken there?

Neil Ferguson: *[00:24:49]*
Well, I mean, Bolton in the north of England has been highlighted as a hotspot and it was a hotspot earlier in the pandemic in terms of transmission last autumn. And it's true that vaccination levels were below the national average up to a few weeks ago. And so, I mean, the local community and the government have responded by what could be called surge vaccination by doubling down on asking people to get vaccinated and providing mobile vaccination centers so that people who previously may have been hesitant about being vaccinated were able to then be vaccinated. And that undoubtedly will help.

Moderatorin: *[00:25:36]*
And Mr. Gupta, do you think that the shortening of the interval also could help to decrease the spread of the virus if now the interval between the two vaccine doses is shortened.

Ravindra Gupta: *[00:25:52]*
Yes, I mean, we have a 12-week interval and no doubt, you know, the ideal thing is to have a three-week gap between the vaccinations so that you are protected earlier to a reasonable level and certainly in the more vulnerable and people with poor immune responses. I think that bringing them closer together is probably advantageous.

Moderatorin: *[00:26:15]*
Before you need to leave now, I would like to ask you a last question and I would like to look a bit in the future. Following SARS-CoV-2 now for almost one and a half years and observing the rise of different variants of concerns like B.1.1.7, P.1 and now the B.1.617.2 strain. What kind of variants do you think we need to prepare for? What could there still be coming?

Ravindra Gupta: *[00:26:42]*
Yeah, that's really important question, I think. We have good vaccines now, we need to keep the pressure on vaccine designers, manufacturers to adapt vaccines and use, and we really need to think carefully about what we do, how we do that, what mutations, what combinations, what adjuvants, what bits of the virus we need to use, is it live viruses, is it mRNA vaccines. These are things that are still open questions for longer term control. Secondly, the virus is going to do some weird things. I mean, this is just the beginning. I think it's going to recombine. You're going to get super mutant viruses, I believe. But that's not necessarily bad. I mean, it's not necessarily a terrible thing, but the virus is going to do very unexpected things because the amount of pressure on it is going to be severe. So, it will adapt, as we all know, and we know that people still get Corona infections and that's how this all happens in general. It's hard to say what's going happen, but the virus is going to find ways of becoming more infectious. You can see that already. When it's under pressure, it will try and be more efficient in transmission so that it can achieve the job with fewer virus particles. And

so, things like the P681R recession are just the beginning. There'll be there'll be further ones coming, I'm sure. So not only antibody escape, but actually increasing transmission advantage or infectivity advantage is next.

Moderatorin: *[00:28:06]*
And would you expect that if it can transmit quicker, but if the population is largely vaccinated, then our societies would still not be overwhelmed, because if they are largely immunized, then it's maybe just like a cold?

Ravindra Gupta: *[00:28:28]*
I think as the coverage goes up, yes, for most people it will be a mild illness, even with these super variants or whatever you are going to call them. Most people will still be protected against the severe effects of the disease. So that's great. But there will be some vulnerable people that we see with flu. We have a lot of deaths each year from flu in vulnerable groups. We try to vaccinate them first to protect them. But it doesn't always work. But I don't think we should say it's going to be like flu automatically. I think that this is an unpredictable virus, and we shouldn't be overconfident at any stage.

Moderatorin: *[00:29:04]*
All right. And then the next question to you, Mr. Ferguson. The current reopening's that we see across European countries, could they help the spread of the Indian variant, and how inevitable is it that this variant will become dominant like the Kent variant did a few months ago?

Neil Ferguson: *[00:29:32]*
So, actually, reopening's are going to help any variant spread. If you have high contact rates between people, that's good for the virus. Perhaps, it's probably likely that the B.1.617.2 will dominate but not certain at the moment. We just need more information on really the level of transmission advantage. If it is substantially more transmissible than B.1.1.7 it will eventually dominate.

Moderatorin: *[00:30:03]*
And how should stronger measures against the spread of the variant be balanced with the success of the vaccine campaigns?

Neil Ferguson: *[00:30:13]*
So, I mean, we had an earlier question on this topic. I think, it's not an all or nothing thing. All of these key quantities are a matter of degree: How much does this variant manage to evade vaccine induced immunity? How much more transmissible is it will determine really whether any future third wave in the U.K., for instance, in the summer or early autumn is manageable or risks overwhelming the health system again, and therefore a reversal of the current U.K. road map out of lockdown. And so, the road map the U.K. is adopting in the context of a high level of vaccine coverage and gradually reopening is robust to a certain level of increasing transmissibility of the virus and a certain limited level of immune escape, of evading the vaccines, but only a certain amount. If it goes beyond those levels, then we need to reconsider

the rate of reopening and maybe slow the next step. I know less about all the different plans in every European country, but the same principles will hold. I mean, if one behaves cautiously and reopens gradually and evaluates the data step by step on what every step and relaxation does to transmission, then you're much more likely not to need to have to reverse course later.

Moderatorin: *[00:31:49]*
Experts in the U.K., that also include your group, have suggested a more transmissible virus could cause a large third wave of hospitalizations even without the modest vaccine escape shown. Does this still hold? You sound more optimistic now.

Neil Ferguson: *[00:32:08]*
Well, I say, it's a matter of degree. If you hypothesize and look at a scenario where a variant is, let's say, 60 percent more transmissible, has a certain degree of immune escape, that could well lead to another third wave the size the U.K. has just come out of. But if the level of transmissibility increases only 20 percent or 30 percent and there's only a little bit of evasion of immunity, then you get a much, much lower third wave. There's not … a direct linear relationship between the extent of transmission advantage and the size of any predicted third wave. It's a much more complicated situation than that. It's almost akin to having a threshold. We can cope with a certain level of increased transmissibility and still be in a good position to continue with the road map. But if it's higher than that, we need to reconsider.

Moderatorin: *[00:33:09]*
There is a follow up question on the topic that we touched before, infections in younger people. And one journalist is asking this in addition. He wants to check if you are referring to B.1.617.2 when we comment on infections under 21 and younger people.

Neil Ferguson: *[00:33:33]*
Yes, just to reiterate, what we see is: There's a slightly greater propensity to B.1.617.2 infections to be affecting those under 21 than B.1.1.7. But I just want to reiterate, that may have nothing to do with the biological properties of the virus. It may just be a factor of the communities within which this infection has been seeded from travelers from India.

Moderatorin: *[00:34:03]*
OK, I would like also now to come to an end, and with you I would like also to look into the future and how you evaluate the next months. The next opening steps are coming in June, I think. And how do you look in the future? How do you evaluate what will come?

Neil Ferguson: *[00:34:28]*
So, if I could completely predict the future, I mean, I would be in a much better position. I think we're continuing to evaluate data. I think it's actually too early to say whether we will be able to go ahead with what was planned in the U.K. in mid-June and the next step, basically a full relaxation of measures, or whether that fourth stage of relaxation will need to be postponed or indeed, in the worst case, measures need to be tightened up. We're getting more and more

data every week, but we hope to be in a position to be more definitive about these answers in the next two to three weeks.

Moderatorin: *[00:35:07]*
From what you've known from the B.1.1.7 development in the beginning of the year, what needs to happen now that you would tell the responsible persons to really stop opening up and increase the restrictions again, what needs to happen? How high do the cases have to rise or what do you have to see in the population?

Neil Ferguson: *[00:35:38]*
(...) It's not about how high cases rise. It's about how quickly they're rising. And in particular, are we seeing evidence of a rapid rise in hospitalizations. I mean, at the moment, we're at very low infection levels, very low numbers of people hospitalized. But it's the trend which is important. If we start seeing, for instance, case numbers doubling every 10 to 14 days and hospitalizations following the same track, that would be of concern. We always expected to see case numbers rise as we relax, and that's sort of built into the plan that we can cope with that. It's just if they are rising too quickly, and that will be a problem.

Moderatorin: *[00:36:17]*
But there just came in another question that I quickly forward to you. Which data do you await with modelling the impact of new variants? Which ones are you missing to better assess the future?

Neil Ferguson: *[00:36:32]*
(...) I mean, the U.K. has a remarkable amount of data coming in, both genetic sequence data, which is the most important probably for variants, but we link that data to things like case symptomatic, case testing, to immunization records, to hospitalization records. On top of that, we have data streams like serology in the population, the overall numbers vaccinated. And it's all at a very fine individual level. So, it's pulling all of those different data sets together and linking them, which gives us insight so quickly into things like what impact is this variant having on vaccine effectiveness.

Moderatorin: *[00:37:15]*
Mr. Ferguson, thank you so much for taking the time. For you out there, I would like to remind you that we recorded this briefing, and you can watch it again by entering our website. And we also will transcribe the briefing, and you will find the transcript as soon as possible – also on our website. Thank you so much for your attendance. Mr. Ferguson, thank you so much for your time. Have a good afternoon, bye.

Ansprechpartnerin in der Redaktion

Annegret Burkert
Redakteurin für Medizin und Lebenswissenschaften

Telefon +49 221 8888 25-0
E-Mail redaktion@sciencemediacenter.de

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