Witness Name: Professor Sunetra Gupta
Statement No.: 1
Exhibits:
Dated:

UK COVID-19 INQUIRY

WITNESS STATEMENT OF PROFESSOR SUNETRA GUPTA

I, Professor Sunetra Gupta, will say as follows: -

My expertise

1. I am Professor of Theoretical Epidemiology at the University of Oxford, with an interest in a range of viral, bacterial and parasitic infectious diseases such as malaria, HIV, influenza, meningitis and pneumonia.

2. I have a substantial track record in the application of theory to public health and therefore was well-placed to comment upon government policies concerning lockdowns and other interventions aimed at reducing the spread of infection. I was - and continue - to be critical of the idea that a national lockdown is the right strategy response to the SARS-CoV-2 pandemic, and have instead advocated a strategy of 'Focused Protection' of those vulnerable to severe disease and death. Although my advice was generally disregarded at the time, it is now becoming accepted practice in the face of gathered evidence on the range of lockdown responses and the poor performance of competing strategies.

3. With regard to expressing my views on the SARS-CoV-2 pandemic, I have published numerous articles in the mainstream media and been interviewed extensively by a wide range of media outlets and several public forums. Despite being the subject of much derision and abuse, these ideas are now being accepted

as a viable alternative to measures which entail a high degree of collateral damage, particularly in the developing world and to children, worldwide.

4. In addition to epidemiology, I also have expertise in immunology and vaccinology. Under my direction, my research group are now using the body of theoretical work on pathogen evolution I have consolidated over the last three decades to identify novel methods of designing vaccines against many important diseases caused by pathogens that are capable of mutating to avoid immunity. We have successfully patented our approach against a major subtype of influenza (H1); this technology has been licensed by Blue Water Vaccines Inc. who were set up expressly for this purpose, and have now listed on the New York Stock exchange (NASDAQ:BWV). This work presents, as far as I know, a unique example of a mathematical model of the evolutionary dynamics of an infectious disease leading to the experimental identification of a novel vaccine target.

5. My group's expertise in immunology allowed us to be among the first to develop an assay to measure functional antibody levels to SARS-CoV-2 in the interests of ascertaining the extent of spread, as well as to study the immunological relationships between SARS-CoV-2 and endemic coronaviruses. We have used mathematical models to advance hypotheses concerning the influence of previous exposure to endemic coronaviruses on the outcome of SARS-CoV-2 infection which are currently standing scrutiny rather better than alternative conjectures. The relevant papers may be found among my peer-reviewed publications.

6. I am a founder member and trustee of Collateral Global, a UK registered Charity (No. 1195125) which is dedicated to researching, understanding, and communicating the effectiveness and collateral impacts of the Mandated Non-Pharmaceutical Interventions (MNPIs) taken by governments worldwide in response to the COVID-19 pandemic. Through this vehicle, I have produced a set of mini-lectures for the public to make accessible some of the fundamental principles of infectious disease. This synergises with my strong interest in public engagement and promotion of women and minorities in science.

My scientific position on the dynamics of the SARS-CoV-2 pandemic

7. The observed dynamics of SARS-CoV-2 (i.e. how the virus spreads) in most global settings can be explained by assuming that the virus arrived at different times within the seasonal cycle of changes in viral transmissibility and – as is the case with seasonal coronaviruses – that immunity from natural exposure does not durably block infection, even though protection from severe disease can be lifelong.

8. Within such a framework, the acute phase of the pandemic would have occurred prior to March 2020 (leading to the wave of deaths in April 2020) in the UK and sufficient immunity would have accumulated in the population to keep infections at low levels over the summer. As anticipated by many of us who attempted to put together a strategy to deal with this eventuality, a second wave occurred in the autumn when the proportion immune again fell below the herd immunity threshold.

9. The alternative explanation is that these patterns arose as a result of lockdowns and other non-pharmaceutical interventions rather than herd immunity. Under this scenario, the lifting of restrictions should have caused a very large increase in infections. That this did not happen allows us to dismiss the conjecture that NPIs had any significant effect on community spread.

10. But isn't it just the case that the virus has evolved into a milder but more transmissible form? We must be very careful about making such an assumption, not least because it suggests that how we handle the situation now should be different from how we handled it in the first place.

11. The omicron variant is capable of significant immune evasion – this gives it an edge over delta in re-infecting those who have either been previously exposed or vaccinated. This is why it has replaced delta. We see this happening all the time with influenza. It does not require any increase in transmissibility. While some very elegant studies have shown that the omicron variant has some clear functional differences from its predecessors, there is no reason to believe it is

intrinsically less virulent or more transmissible. The idea that all viruses evolve in this direction is entirely incorrect.

12. It would have been most unreasonable to expect SAGE modellers to recalibrate their models on the basis that omicron was less virulent. Instead, they assumed – correctly, in my opinion – that the reason that omicron was so 'mild' in South Africa was because of high levels of previous exposure. Their predictions thus made complete sense under their assumption that the levels of natural exposure in the UK were low. However, it is this assumption that was likely flawed, rather than any dereliction of duty on their part to 'update' their model to reflect that omicron was intrinsically less severe.

13. In summary, SARS-CoV-2 has behaved as any standard epidemiology textbook, coupled with a passing acquaintance with the characteristics of other seasonal coronaviruses, would lead you to expect. It has achieved its predicted endemic state; its dynamics are determined by the waxing and waning of natural immunity against a background of seasonality in transmission; it was never any more virulent than the other seasonal coronaviruses (only the vulnerable were especially at risk because they had never encountered SARS-CoV-2 before); and it evolved to evade natural immunity or to marginally improve transmissibility (which is all it needed to outcompete the prevailing variant).

My position on the management of the SARS-CoV-2 pandemic

14. Making difficult decisions in the face of uncertainty is a common feature of all crises. The SARS-CoV-2 pandemic was no exception; but, sadly, much of the decision-making was conducted under a misapprehension of the uncertainties involved.

15. What were these uncertainties?

    a. Uncertainties concerning the dynamics of SARS-CoV-2: As explained above, these were amplified beyond what any rational scientific

assessment of the situation would suggest, given how much we already knew about coronaviruses.

b. Uncertainty of the extent to which non-pharmaceutical interventions would curb the spread of infection, and to what end: This was downplayed, and those who dared to question their efficacy were treated as heretics.

c. Uncertainty of collateral damage: Given the conditions under which the majority of the world's population lives, if there was one thing we could be sure of it was that people would die—of hunger, malnutrition, disease and malaise—as a result of lockdowns. This has now been painfully corroborated, as has the effect on more affluent nations where much of the significant social, economic, psychological and developmental harms have been suffered by the poor and the young.

16. In effect, we betrayed the Precautionary Principle by adopting a strategy which was certain to cause harm but whose benefits were entirely unclear.

17. Much of the ongoing confusion about what NPIs can achieve has arisen from the reluctance to distinguish between the benefits and costs of particular NPIs at an individual level, at the level of an institution (eg a hospital or care home) and within the population. The distinction between how these measures affect the individual, the institution and the population is crucial, as I will explain below.

18. We need to move away from the binaries of pro- and anti-lockdown and consider instead exactly what the NPIs in question delivered at each of these levels in terms of (i) reduction of SARS-CoV-2 infection, (ii) reduction of severe Covid illness (iii) reduction of infection with other endemic pathogens; these then must be weighed up against their other costs and benefits.

19. At an individual level, certain NPIs – e.g. social isolation - do protect against infection; however, these same interventions have a negligible effect on the spread of an epidemic in the population unless implemented in an extreme manner which is simply not sustainable. Certain NPIs can also be used to prevent the spread within an institution (eg. a hospital or care home). NPIs can be used (in extreme form) to keep the virus from entering a population, but this option is only available

to specific, generally remote, locations. Within a framework of global co-ordination, we may well have agreed that New Zealand should shut its borders while a vaccine was being developed to protect the vulnerable and they, in turn, would perhaps provide the facilities for conducting a trial, for example; instead, we had to suffer their mindless self-congratulation in “keeping the virus out”.

20. NPIs will have a negligible effect on the reduction in hospitalisable Covid illness at an individual level, since the IFR is so low for the majority of the population; however, it is a powerful means of protecting the vulnerable population. Reduction of severe disease in institutions (e.g. a hospital or care home) and at population level will only be achieved if the vulnerable population is adequately shielded. Trying to achieve this by attempting to stop the spread is tantamount to letting it rip.

21. With regard to the costs and benefits of measures such as lockdowns, at an individual level, this will vary with socio-economic status and disproportionately harm the poor and the young. At a population level, severe economic losses will be sustained which will in turn also have devastating effects on the poor and the young. Institutions such as hospitals maybe seriously constrained in their delivery of healthcare, ultimately leading to large numbers of deaths; care homes are likely to see a massive decline in the quality of life for those at the end of their lives.

22. It is possible to capitalise on the ability of NPIs to protect (vulnerable) individuals from severe disease, while avoiding the costs for the rest (i.e. majority) of the population and harm done by NPIs to the healthcare system, through a system of Focused Protection.

23. Focused Protection is robust to the uncertainties regarding the ability of NPIs to curb the spread of infection at the population level and is predicated on the certainty that these interventions will cause extreme harm.

24. My view is that the logic of what I have outlined above was both available and should have been obvious to those whose responsibility it was to manage the response to the pandemic, and in fact many of these arguments had been formed

and were known to professionals in the field well before 2020 as part of national pandemic planning. The blind adoption of lockdown and lack of debate as to how to respond to the uncertainties is a tragedy for which the whole of society is now paying a hefty price.

My specific involvement (as such) in the decision-making process

25. Since 2001, when I published an opinion piece on the subject in Nature (*exhibit SG/01 - INQ000000000*), I have been increasingly concerned at the use of mathematical modelling as a predictive tool. Models are extremely useful in providing a framework for scientists to develop testable hypotheses but can be dangerously misleading when used to make predictions, particularly when the complexity of the model obfuscates key assumptions.

26. In March 2020, my colleagues and I released a pre-print (*exhibit SG/02 - INQ000000000*) demonstrating the wide uncertainty that existed within estimates of expected numbers of deaths by fitting a mathematical model to the available data. We showed that the available data were equally compatible with a very low IFR in an epidemic whose first peak had occurred much earlier in the year as with the much higher IFR in the Ferguson et al model which was predicated on a much more limited spread of infection. We emphasized the importance of conducting studies which would reveal the true extent of the spread of infection before coming to conclusions about the lethality of SARS-CoV-2 in vulnerable individuals. It was already clear at that time that the majority of the population were at very low risk of severe disease and death, and therefore substantial spread could easily have occurred without being registered as a clinical phenomenon; there were also some syndromic data which suggested that there were certain foci in which many individuals had suffered a Covid-like illness.

27. Members of SAGE reacted both publicly and privately to this paper by acknowledging the validity of its primary findings of uncertainty concerning true extent of spread (Vallance said so in daily briefing, Ferguson during questioning by parliamentary committee). In an amicable conversation with Ferguson, he and

I agreed that serological surveys were needed to assess this – although he was fairly sure that it had not spread much at all in the UK.

28. As already mentioned, my team was among the first to develop a functional antibody assay to measure SARS-CoV-2 exposure. We were however prevented from obtaining any samples, other than from blood donors in Scotland; our analyses of these indicated that some spread had occurred in Glasgow (we were able to track an increase in seropositivity through late March into early April) but that most of the rest of Scotland had very low levels of infection. These results do not contradict the assertion that the decline in infections in March 2020 occurred through a build-up of herd immunity and a seasonal decline in transmissibility. Several regions had indeed not yet experienced the full force of the pandemic but many urban areas, in particular, had sustained high rates of exposure. This is clear in the signatures of serologic studies, once it is taken into account that the antibodies being measured only have a half-life of a couple of months.

29. The levels of exposure required to achieve herd immunity (i.e. decelerate infection rates) are also complicated by previous exposure to seasonal coronaviruses. We released a pre-print (*exhibit SG/03 - INQ000000000*) to elaborate on this phenomenon, but it was ignored and derided. Data has since been mounting in support of our hypothesis; the protection offered by other circulating coronaviruses also explains why seroconversion rates to SARS-CoV-2 may have been lower than expected in certain regions.

30. Due to these various uncertainties, it was not possible to predict the magnitude of the imminent second wave and therefore it was important to formulate a strategy that was robust to uncertainty. Focused Protection of the vulnerable population offered what appeared to me to be the only humanitarian route out of the crisis, and I expressed this view (*exhibit SG/04 - INQ000000000*) in the Cabinet meeting I was invited to attend via Zoom with Carl Heneghan, Anders Tegnell, John Edmunds and Angela McLean. Each of us provided a statement: mine and Professor Heneghan's are available in *exhibit SG/04 - INQ000000000*. Anders Tegnell gave a general outline of the "Swedish" strategy which corresponded to the opinions he had already expressed on multiple occasions in the press; Angela

McLean expressed the opinion that we should be doing whatever Tegnell was doing. As the "Swedish" strategy is effectively synonymous with focused protection of the vulnerable, it could be said that other than John Edmunds, all invitees were broadly in favour of proposals outlined by Tegnell. Boris Johnson interrogated each of us on our position but there was no opportunity for a panel discussion. Other than Rishi Sunak, I was unable to specifically identify who was there in the room.

31. The following week, an open letter was published by myself and Profs Carl Heneghan and Karol Sikora (with 23 other signatories) with "the intention of providing constructive input into the choices with respect to the COVID-19 policy response". I was subsequently contacted by academics in USA (with none of whom I had any previous professional or personal contact) who were also keen to open up the discussion to consider alternatives to lockdown and met with Profs. Martin Kulldorff and Jay Bhattacharya (we were joined remotely by Dr. Stefan Baral) at a press conference in Great Barrington, Massachusetts. The so-called "Great Barrington Declaration" [*exhibit SG/05 - INQ000000000*] arose out of our deliberations and was published on October 4, 2020. It is entirely incidental that this occurred on the premises of the American Institute for Economic Affairs; we were not in their pay, nor were we driven by any alignment with their politics (I am a strong advocate of the welfare state); they merely offered a venue. On October 6th, Profs. Bhattacharya, Kuldorff and myself met in Washington DC with United States Secretary of Health and Human Services (Alex Azar) and outlined our position to him. We also met with advisor on the White House Coronavirus Task Force, Professor Scott Atlas, who shared many of our views on the impacts of lockdowns to the poor and the young.

32. Following this, I and many of the academics named above became victims of a vicious campaign to silence and impugn us. Many of my energies have been consumed by resisting these accusations and dealing with the distress caused to myself and my family. We were branded as right-wing extremists, in the pay of the Koch Brothers. The MP Neil O'Brien and others established a website (*exhibit SG/06 - INQ000000000*) to attack us specifically. Many seized on a statement offered to Freddie Sayers, editor of UnHerd, in May 2020 that the epidemic was

on its way out; while it may have been awkwardly worded, it certainly was not my intention to convey that infection levels would never rise again. If I had truly believed this then I would have hardly wasted my time formulating a plan for the winter of 20/21 on the basis that there would be a second wave.

33. At this time, another group of academics came out with a document entitled the 'John Snow Memorandum' in which they suggested, rather remarkably, that we may never reach herd immunity given that SARS-CoV-2 was unlikely to induce long-lived infection blocking immunity upon infection. This is entirely untrue and, as I have explained before, much of the journey towards the endemic equilibrium state (where the levels of immunity in the population hover around the herd immunity threshold) had been accomplished in many areas of the UK during the first wave.

34. Much more reasonably, many questioned how Focused Protection might actually be delivered – but instead of engaging in serious discussion and debate around this point, we were ridiculed for daring to make this suggestion. It remains my opinion that we could have protected the vulnerable at a fraction of the expenditure on lockdowns while avoiding the enormous human costs.

35. I have had some positive interactions with other MPs: (i) I met with Theresa May on 28/10/20 as she wished to interrogate me on my views regarding the necessity of lockdowns, (ii) I had conversations with Graham Brady and Stephen Baker in September 2020 along the same lines. I have had no further direct involvement with the Cabinet. Shortly after the meeting in September 2020, Michael Gove expressed an interest in speaking with me concerning my views on lockdowns but repeatedly cancelled. I have no idea why the meeting did not materialize.

36. Since the beginning of 2021, I have focused my attention on raising awareness of the harms of lockdowns through Collateral Global as well as attempting to educate the public - and some in public life - on the simple principles of infectious disease epidemiology.

Recommendations for the Inquiry

37. Should we have locked down earlier? For many, examining our level of preparedness is synonymous with the timing and effectiveness of the non-pharmaceutical interventions (NPIs) employed to stop the spread of the virus in the population. And yet, there is almost no evidence to support that such measures achieved the same; indeed, the data would appear to indicate that the majority of these NPIs did very little to halt the spread of the epidemic. Instead, they caused endemic diseases, such as influenza, to disappear transiently (as would be predicted by well-established epidemiological theory), thereby exposing us to the undesirable consequences of an immunity debt towards these other problematic pathogens.

38. Focus on the key trade-off. Rather than wasting time and energy on understanding why we were not more eager to implement such measures earlier in the epidemic, I believe a more useful line for the Covid Inquiry would be to examine why we did not distinguish between on the one hand the benefits the measures could have brought at an individual level to those at risk of severe disease and on the other the harms they were bound to cause at a population level, especially for the poor and the young.

39. Examine the effect of the measures taken on the institutions they were designed to protect. We should also ask why we adopted NPIs to prevent community spread – which effectively led to the collapse of the institutions, such as the NHS, that these measures were designed to protect - instead of using NPIs to prevent the spread of the virus to vulnerable individuals within healthcare settings and care homes.

40. Examine the role of the media. We need to question the role of both mainstream and social media, scientific journals and institutions in shutting down and censoring crucial debate.

Statement of Truth

I believe that the facts stated in this witness statement are true. I understand that proceedings may be brought against anyone who makes, or causes to be made, a false statement in a document verified by a statement of truth without an honest belief of its truth.

Signed: _____Sunetra Gupta_____

Dated: _____25/8/23_____