THE SPECTATOR

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We know everything – and nothing – about Covid

It is data, not modelling, that we need now

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We know everything about Sars-CoV-2 and nothing about it. We can read every one of the (on average) 29,903 letters in its genome and know exactly how its 15 genes are transcribed into instructions to make which proteins. But we cannot figure out how it is spreading in enough detail to tell which parts of the lockdown of society are necessary and which are futile.

Several months into the crisis we are still groping through a fog of ignorance and making mistakes. There is no such thing as 'the science'.

This is not surprising or shameful; ignorance is the natural state of things. Every new disease is different and its epidemiology becomes clear only gradually and in

new disease is different and its epidemiology becomes clear only gradually and in retrospect. Is Covid-19 transmitted mainly by breath or by touching? Do children pass it on without getting sick? Why is it so much worse in Britain than Japan? Why are obese people especially at risk? How many people have had it? Are ventilators useless after all? Why is it not exploding in India and Africa? Will there be a second wave? We do not begin to have answers to these questions.

As a result, we don't really know what works. It is possible that washing your hands, not shaking hands with others, not gathering in large crowds, and wearing a face mask in public, but no more than this, might have been enough, as Sweden seems to suggest. Forcibly shutting schools and shops and aggressively policing sunbathers in parks may have added little in terms of reducing the rate of spread.

There is one vital fact that emerges from the fog. Countries that did a lot of testing from the start have fared much better than countries that did little testing. This is true not just of many Asian countries, such as South Korea (though Japan is an exception), but within Europe too. Up to the middle of last month, Iceland, Lithuania, Estonia and Germany had done many more tests per million people and recorded many fewer deaths per million people than Belgium, Britain, Italy and Sweden. As Max Roser of the website Our World In Data puts it: 'The countries with the highest death rates got there by having the lowest testing rates.'

This is true of regions within countries, too. In Groningen in northern Holland the overall death rate is up just 4 per cent on last year, while in North Brabant, in the south of the country, the death rate has doubled. The difference is down to testing: on 19 March Professor Alex Friedrich insisted on Groningen refusing to adopt the new national policy of testing only severe and priority cases. Groningen continued testing as many people as possible. Britain's failure to ramp up testing in mid-March — and to limit testing to those already in hospital with symptoms — is its biggest mistake, not its failure to lock down the economy sooner.

Yet it is not obvious why testing would make a difference, especially to the death rate. Testing does not cure the disease. Germany's strange achievement of a consistently low case fatality rate seems baffling — until you think through where

most early cases were found: in hospitals. By doing a lot more testing, countries like Germany might have partly kept the virus from spreading within the healthcare system. Germany, Japan and Hong Kong had different and more effective protocols in place from day one to prevent the virus spreading within care homes and

hospitals.

The horrible truth is that it now looks like in many of the early cases, the disease was probably caught in hospitals and doctors' surgeries. That is where the virus kept returning, in the lungs of sick people, and that is where the next person often caught it, including plenty of healthcare workers. Many of these may not have realised they had it, or thought they had a mild cold. They then gave it to yet more elderly patients who were in hospital for other reasons, some of whom were sent back to care homes when the National Health Service made space on the wards for the expected wave of coronavirus patients.

The evidence from both Wuhan and Italy suggests that it was in healthcare settings, among the elderly and frail, that the epidemic was first amplified. But the Chinese authorities were then careful to quarantine those who tested positive in special facilities, keeping them away from the hospitals, and this may have been crucial. In Britain, the data shows that the vast majority of people in hospital with Covid-19 at every stage have been 'inpatients newly diagnosed'; relatively few were 'confirmed at the time of admission'. The assumption has been that most of the first group had been admitted on an earlier day with Covid symptoms. But maybe a lot of them had come to hospital with something else and then got the virus.

Even if you combine both groups, there are hardly enough admissions to explain the number of deaths in hospitals, unless nearly everybody admitted to hospital with Covid has died. It is likely that the frail and elderly, which the virus singles out for punishment, were more likely to be going to hospitals or clinics for other ailments and it was there that many of them got infected during February and March.

In Belgium, the country with the worst epidemic per head of population (though the numbers are inflated by the way the country defines a Covid death), all 210,000 people in care homes, both residents and staff, were tested in the second half of April. Some 10 per cent of them tested positive for the virus. That's actively having it at the time of the test, not having had it: one in ten!

If Covid-19 is at least partly a 'nosocomial' (hospital-acquired) disease, then the pandemic might burn itself out quicker than expected. The death rate here peaked on 8 April, just two weeks after lockdown began, which is surprisingly early given that it is usually at least four weeks after infection that people die if they die. But it

makes sense if this was the fading of the initial, hospital--acquired wave. If you look at the per capita numbers for different countries in Europe, they all show a dampening of the rate of growth earlier than you would expect from the lockdowns.

This idea could be wrong, of course: as I keep saying, we just don't know enough. But if it is right, it drives a coach and horses through the assumptions of the Imperial College model, on which policy decisions were hung. The famous 'R' (R0 at the start), or reproductive rate of the virus, could have been very high in hospitals and care homes, and much lower in the community. It makes no sense to talk of a single number for the whole of society. The simplistic Imperial College model, which spread around the world like a virus, should be buried. It is data, not modelling, that we need now.

A study of 391 cases of Covid-19 and 1,286 of their contacts, in the Shenzhen region of China, found that 80 per cent of cases were transmitted by just 9 per cent of carriers, and that only 11 per cent of those sharing a household with a case caught the virus. By contrast, a study of a nursing home in Washington state found that 23 days after the first case was diagnosed on 20 January, 64 per cent of residents tested positive, half of them showing no symptoms. An analysis by Dr Muge Cevik of St Andrews University of 14 similar studies concluded that prolonged and close contact is necessary for transmitting the virus and the risk is highest in enclosed environments: households, long-term care facilities and public transport. She adds: 'Casual, short interactions are not the main driver… Epidemic intensity is strongly shaped by crowding.'

If the elderly, obese and frail are not just at greater risk of dying, but also more susceptible and more infectious, then by definition everybody else is less so. Gabriela Gomes and colleagues at the Liverpool School of Tropical Medicine looked at what would happen if the susceptibility of different segments of the population to the virus is very different, and concluded that in some circumstances effective herd immunity could be achieved with as little as 10 per cent of the population immunised. In the words of the study: 'Individuals that are frailer, and therefore

more susceptible or more exposed, have higher probabilities of being infected, depleting the susceptible subpopulation of those who are at higher risk of infection, and thus intensifying the deceleration in occurrence of new cases.'

If this is right, then it is good news. Once the epidemic is under control in hospitals

If this is right, then it is good news. Once the epidemic is under control in hospitals and care homes, the disease might die out anyway, even without lockdown. In sharp contrast to the pattern among the elderly, children do not transmit the virus much if at all. A recent review by paediatricians could not find a single case of a child passing the disease on and said the evidence 'consistently demonstrates reduced infection and infectivity of children in the transmission chain'. One boy who caught it while skiing failed to give it to 170 contacts, but he also had both flu and a cold, which he donated to two siblings. Children appear to have ACE2 receptors, the cellular lock that the coronavirus picks, in their noses but not their lungs.

This makes models based on flu, a disease that hits the young hard, misleading. The more the coronavirus has to use younger people to get around, the weaker its chances of surviving. Summer sunlight should slow it further, both by killing the virus directly and by boosting vitamin D levels. Vitamin D protects against colds and flu, and especially at the end of winter is often deficient in obese, dark-skinned or elderly people, all of whom have proved more susceptible to Covid-19. In a study in Indonesia, Covid-19 cases with deficient vitamin D were an enormous 19 times more likely to die from the disease than people with adequate levels.

It won't be straightforward and there will be setbacks, but testing, followed by track and trace, is plainly now the way out. Britain is belatedly catching up. Matt Hancock's ambitious dare to the healthcare system to get to 100,000 tests a day had the desired effect. We are now brimming with testing capacity, albeit still too centralised and slow in getting results back to people. The Office for National Statistics is starting to gather data that will give a national picture. The antibody test is coming. So are apps to tell us if we have come close to an infected person. Precautions like face masks will now become widespread. It would be a surprise, given that the virus is not very good at spreading among younger people, if it could survive such an assault on more than one front.

Then we have to tackle another set of unknowns relating to a different species of creature: the human being. How people react to an easing of the lockdown is also uncertain. The British government took the paternalist view that we could not be trusted to take advice but must be ordered into lockdown. It rushed through some terrifyingly illiberal legislation. With a few exceptions, the British people appear to have become willing, even censorious, assistants in the enforcement of the rules. The problem is not now people disobeying the rules, but being terrified to give up the extreme safety of lockdown and relaxed about staying at home on taxpayer--

subsidised wages. In the light of what we know, it is vital that the government now switches from urging us to stay at home to urging us to return to as much of normal life as possible.

Be in no doubt that the strangulation that is asphyxiating the economy will have to be gradually lifted long before we know the full epidemiology of the virus. Perilous though the path is, we cannot wait for the fog to lift before we start down the mountain.

Now hear the analysis on the Prime Minister's latest address from Katy Balls, ***James Forsyth*** *and* ***Fraser Nelson*** *on the Coffee House Shots podcast:*

spectator.co.uk/podcast *- Matt Ridley and virologist Elisabetta Groppelli on taming the coronavirus. Matt Ridley's How Innovation Works is published by 4th Estate next month.*

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