1 Appendix: Modelling total Covid infections

In the UK, as noted above, we now have ONS surveys of those currently infected (about 2%) and of those with antibodies specific to Covid-19 (about 7%). But in addition an online survey running since end March has recorded those with symptoms of Covid-19; to date it has found a cumulative total of 60% of the population reporting symptoms, mostly fairly weak ones. Total UK deaths have reached 43500, 640 per million — mostly in hospitals and care homes. Recent medical research finds that only those with serious infections develop specific antibodies, while there is general immunity in the population against coronaviruses which can protect against weak infections.

We also find in the latest medical research that those mildly infected, as well as their uninfected family members, develop T-cell responses which are used by the immune system to fight the virus. Thus they have significant immunity to Covid-19, even though they do not develop specific antibodies; furthermore this T-cell immunity is long lasting, over several years, as it seems T-cells are kept in the immune system's memory for long unlike antibodies. At present, the details of this are not clear, awaiting more research. However it now seems that the 60% of the UK population identified by ZOE as having had the infection, have also probably acquired T-cell based immunity against a second wave. If one adds this 60% to the 7% badly infected who acquired antibodies (in addition presumably to T-cells), it suggests that the UK may well now have nearly 70% who have been infected, and who also have long term immunity (i.e against a second wave), implying herd immunity of the basic sort, namely that not requiring any social responses.

How should we take account of this extra data? It would seem that the official PHE etc reported data very greatly underestimates the true extent of infection. Furthermore, the antibody-based infection estimates greatly underestimate those with immunity, especially with long-term immunity (i.e against a second wave). By implication the IFR is much overestimated by the official figures. On the latest figures of deaths vs total so far infected it is 0.1%.

Before the results of the antibody surveys were announced, figures like this were our best estimates based on our modelling and other scraps of data. The antibody tests seemed to destroy them. However, this later data and research suggests they were roughly correct after all. It would seem we are dealing with a population with widely differing pre-existing immunity and resistance to the Covid-19 disease. Many, the vast majority, rapidly developed defences so that they were only weakly symptomatic or even asymptomatic or nearly so. Others, a minority, were badly infected and a proportion of these died.

The data from ZOE records those reporting symptoms of Covid-19 and the ZOE team's estimates of the percent infection rate in the population based on their sample of reports and swab tests taken by reporting people. From these estimates it is possible to estimate daily new cases from the identity $\Delta Infections_t = NewCases_t - RecoveringCases_t$. To obtain the total who have ever been infected at date t we can sum all $NewCases$ up to and including t. Due to recent developments and having access to more data ZOE has recently changed its $Infections$ series from the 11th June. In Figure 1 this change can be seen as a slight drop in the number of people infected. Accumulating the number of people infected we get 40 million as the current number who have been infected — approximately 60% of the population. Adding the 7% estimated to be seriously infected brings the total infected to nearly 70%.

Accordingly, in this Appendix we reestimate our model, benchmarked to a new estimate of the total who have ever been infected; we find a good fit of the model with about 60% or more of the population being predicted to be infected in the long run.

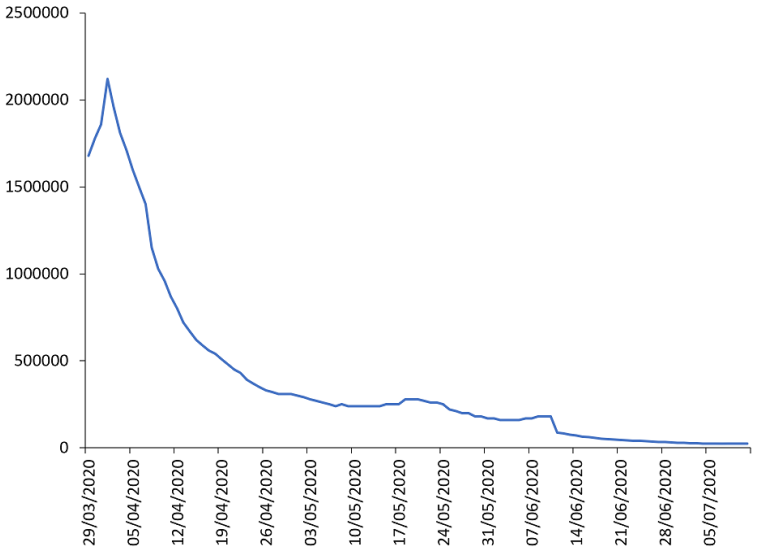


Figure 1: ZOE: number of people calculated to have COVID symptoms

	UK
δ	0.000975
μ	5.14
γ	50.9
ϕ	0.14
$(\mu+\phi)/(\gamma+\phi)$	0.103
Constant	-2.80
% population infected long term	65
Reported/Actual Infections	0.0069
P-value	0.22

Table 1: Structural Model Parameter Estimates

2 Appendix 2: the dynamics of second waves — recent evidence

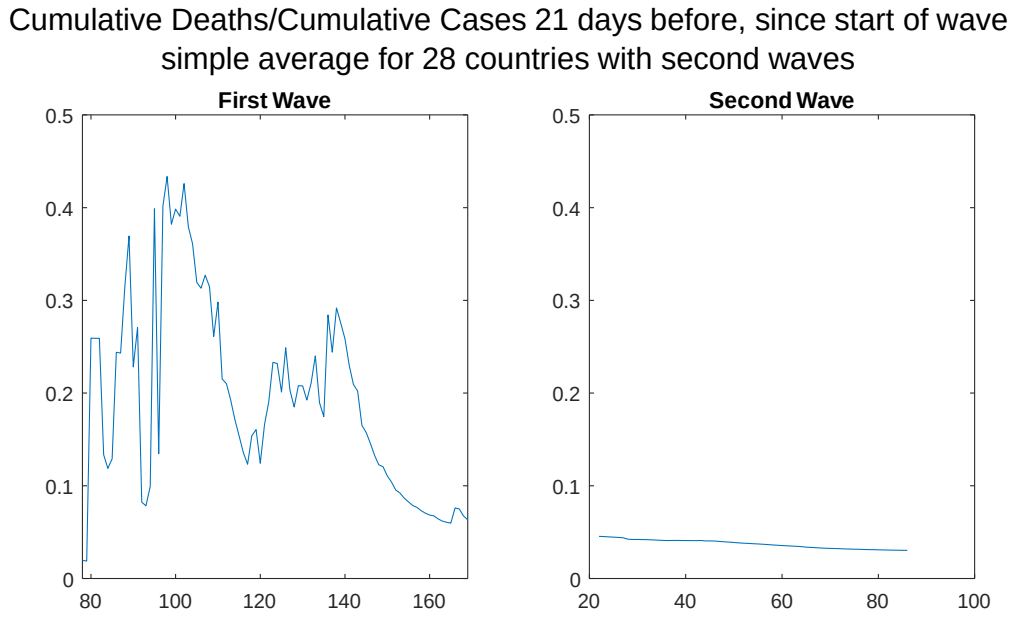
There is now substantial evidence about second waves, since 28 countries have experienced one. We can compare the logistic curve estimates for the two waves, and attempt to find the policy implications for containment of the second.

In these second waves, the same virus has attacked the same population, having already completed a first attack. Evolutionary biology tells us that two main things could have changed between the two waves. First, all organisms come in numerous copies or mutations, from which natural selection weeds out the least fit to survive; hence the surviving virus mutations in the second wave will exclude those that died in the first, whether by killing their host or by being killed by their host recovering from a nasty bout of covid-19 which produced killer antibodies. Second, the population being attacked in the second wave may have more or less immunity than the one attacked in the first; probably more since it will include those who survived from the first wave, while it will no longer sadly include those who did not. So, to sum up it is likely that the virus has 'weakened' and the population has 'strengthened'.

Our 28 countries divide into two groups: 12 with a big second wave relative to the first, and 16 with a small one. The first includes the US, Japan and Vietnam; the second Germany, Netherlands and Singapore. What distinguishes the second group is that it had a longer first wave (tyically four months against three for the other), and seems to have had a good localised test-trace-isolate system operating second time around,

if not also in the first wave.

What all 28 countries have in common is a rapidly falling death rate per reported case. This supports the idea of a weaker virus meeting a stronger population. It could also be due to better treatment, but this had already occurred by the end of the first wave, where the cumulative death rate came down impressively to 10% from a peak of 40%; the fall in the second wave has however been proportionately much bigger, to about 3%. This can be clearly seen in Figure 2, showing the average cumulative death rate for all 28 countries in each wave.



If we use this data to estimate the underlying causes at work, we estimate a much larger 'social reaction' response for the small second wave group than for the other, confirming that they had better policies for 'whack-a-mole'. These we now know involve local area officials providing good local guidance for local behaviour.

Out of the 28 countries, we find sixteen where the second wave has been relatively small — the 'Small' group, while in the other twelve — the 'Large' group — it has been as large or larger than the first. We show the experiences in chart form in Figures 2 and 3[1]. What the first group share is a long-lasting first wave and a policy of localised test-trace-and-isolate after it. All their logistic curves lie below that of the first wave; they all have a lower c, so they reach a lower cumulative total. Mostly they also have a lower b implying that infections peak sooner in the wave: and a lower a, so they fall faster; The second group of twelve tend to the opposite. All their logistic curves lie above that of the first wave; they all have a higher c, so they reach a higher cumulative total. Mostly they have a larger a, so infections fall off more slowly. They also tend to have a lower b implying that infections peak sooner in the second wave.

[1] The lines for the second waves have been set to start at the same figure as the first waves for ease of comparison

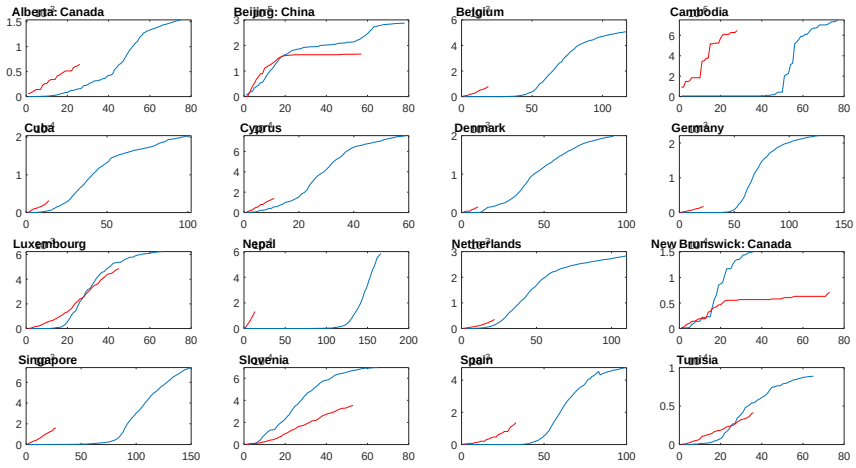


Figure 2: First and Second Wave Comparison (Small Second Wave Countries)

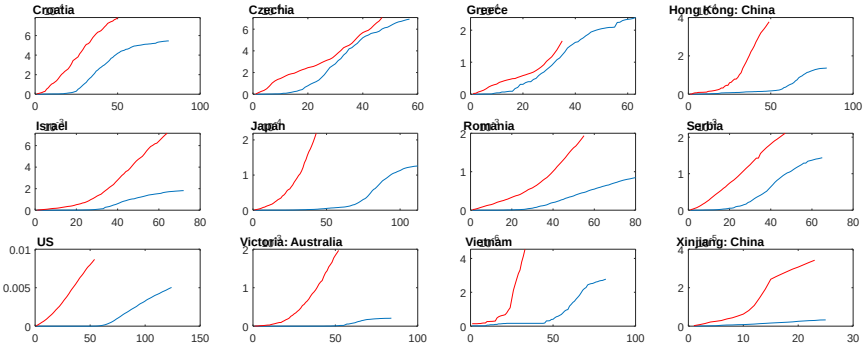


Figure 3: First and Second Wave Comparison (Small Second Wave Countries)

To discover more of what propels these two different second wave trajectories, we estimated a full structural model for the average Small second wave group and the average Large group country, using their average logistic curve behaviour as the matching criterion — the auxiliary model. We estimated their ('augmented') behaviour over the two waves of deaths combined, since the second wave alone furnishes too little data. The data behaviour reveals that the augmented curve for the small group resembles closely that for the first wave alone (see Figure 4) — with only limited extra cases occurring in the second wave: our model estimates for this small group are comparable with typical first wave behaviour in countries with a long infection period — much like the UK with ZOE data — whose estimates we repeat here for convenience. However, for the group with the large second wave, the augmented behaviour is heavily dominated by the large second wave 'tail' (see Figure 5). Accordingly the model estimates for the large group, compared with the small group, weaker government (μ) and social reaction (ϕ) parameters, together with weaker general immunity (γ), while, compared with UK ZOE data, the small second wave group has substantially higher values of all these. The large second wave group behaves quite like the UK ZOE data, apart from having a considerably higher social reaction parameter. In sum, the small group suggests the source of its better results is a combination of more immunity in the population (perhaps from more active sheltering of those more at risk), more goverment reaction and more social reaction.

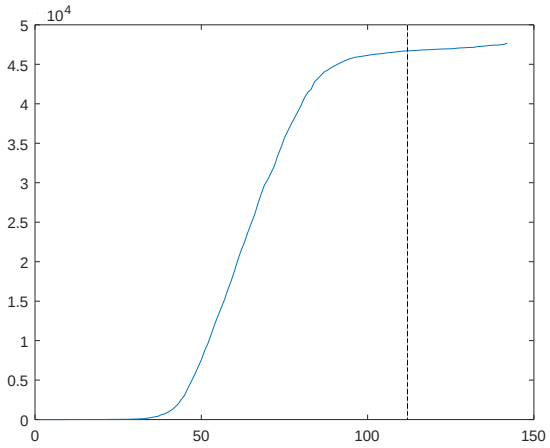


Figure 4: Augmented Small Second Wave Deaths

	UK- ZOE data	Small second wave-aug	Large second wave-aug
δ	0.00975	0.000194	0.00107
μ	5.14	8.5	3.62
γ	50.9	82.7	49.0
ϕ	0.14	31.47	17.15
$(\mu+\phi)/(\gamma+\phi)$	0.103	0.35	0.31
Constant	−2.8	−11.18	−10.27
% Population Infected to Date	7		
% population infected long term	65	75	38
P-value	0.82	0.96	0.17

Table 2: Structural Model Parameter Estimates Comparison

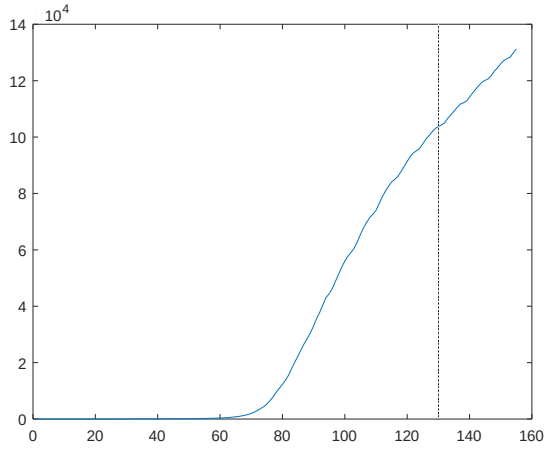


Figure 5: Augmented Large Second Wave Deaths