A structural model of coronavirus behaviour: what do four waves of Covid tell us?

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Abstract

This paper extends Meenagh and Minford (2021) to the four waves of infection in the UK by end-2021, using the unique newly available sample-based estimates of infections created by the ONS. These allow us to estimate the effects on the Covid hospitalisation and fatality rates of vaccination and population immunity due to past infection: the latter was the most significant factor driving both trends, while the vaccination rate also had a significant short run effect on the fatality rate. We also updated our policy comparison with Sweden for the most recent data, with similar conclusions: lower Swedish lockdown intensity relative to personal response in waves 1 and 2 caused much lower economic costs with no discernible effect on infections.

In a previous paper, we set out a structural model of optimising households and biologically-optimised virus behaviour, together with relevant policy interventions, to explain how the Covid virus would spread in the UK and Sweden. We chose Sweden because its policy regime was crucially different and our focus was on how far the UK's more interventionist approach created different results for infections and deaths. In this paper our aim is to study how the four different waves of virus infection the UK and Sweden have experienced have differed over time, so extending our comparative analysis over the full cycle of the pandemic that we have observed. The two countries continued to pursue contrasting policy approaches, allowing us to draw further policy lessons.

Evolutionary biology suggests that viruses evolve to become more transmissible and also less damaging to health, implying a lower fatality rate, because both these developments should increase their survival chances. If this is the case, we should find that across the four waves, the rate of transmission has increased and the death rate per case has fallen independently of the progress in vaccination, which was rolled out rapidly before and during the third wave in the UK, and a little more slowly in Sweden. We already have evidence from the case data that transmissibility increased with each successive variant: in the UK second wave this evidence indicated that the 'Kent variant' dominant in the second wave was 50% more transmissible than the original (first wave) virus; and that the D-variant dominant in the UK's third wave was 50–60% more transmissible again than the Kent variant. However, evidence on the UK fatality rate has been harder to find, partly because NHS-estimated case numbers have been affected by the extent of testing. In this paper we have used ONS-estimates of infections which are based not on those taking tests but on a fixed sampling basis; we have then combined these from their starting point in May 2020 with the NHS data before that, combined with ZOE data on self-reported symptoms, in order to create a full data set across all four UK waves. This early data records those falling ill rather than those taking tests and it should therefore be free of testing bias. For Sweden we have had access only to Johns Hopkins data which comes from Swedish health sources and is for reported cases; since this case data will be biased by reporting processes and we have no sample-based data, we have constructed an infections series from the data on deaths, using the UK estimates of the infection fatality rate for the variant of the time to find implied infections. In this paper

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we have thus drawn on a full and reasonably reliable set of data, for estimating both structural and reduced form models across the full Covid history for both the UK and Sweden; our aim has been to come up with more reliable estimates of these virus features, as well as the effects of vaccination and other interventions. From this updated set of estimates we draw new policy conclusions.

We proceed as follows. First, we set out a new consistent UK series for those infected by Covid, derived from the ONS weekly sample surveys and interpolated to give daily estimates using the ZOE daily survey of those showing symptoms. Though the latter is a voluntary survey and so not calibrated efficiently to the UK population, it is regularly recalibrated to reflect the ONS sample results and so can be used as a supplementary guide to higher frequency infection. Furthermore, we can use it in its recalibrated form to backtrack the ONS data to the earliest periods of infection before the ONS sample began. This new data gives us a reliable series for infections from the start of the pandemic, with four 'waves' of infection to examine.

Second, we estimate our model on these four waves of infection for the UK, to get estimates of the effects of lockdown, immunity spread and social reaction in line with our first paper. The difference is that we are now using infections data not data on Covid deaths, which before was the only reliable data available. We look for any effects of rising vaccination too on the infection process. As in our earlier paper we use indirect inference, using the logistic function estimates as our auxiliary model.

Third, we estimate relationships in all the waves between infections and hospitalisation and deaths. These are simple lagged 'engineering' relationships, in which we look for a simple lag of around three weeks from infection to deaths, and of a few days from infection to hospitalisation. We expect to see progress across the four waves in terms of falling hospitalisation and death rates, as the disease encounters increasing immunity, better health care, and especially rising vaccination rates.

Next, we repeat this process for Sweden.

Finally, we compare and contrast the two countries' features and draw policy implications.

In the next section we repeat our account of the causal model in which both the virus and households choose optimising strategies. The model is the same as in our earlier paper, except that we now introduce Vaccination, V, defined as the population proportion double-vaccinated, as a factor inhibiting the virus' spread. This appears as modifying the previous parameter A to become AV^{ν}

1 The Model:

Let the virus' utility at the start of the infection be given by:

$$U_V = \sum_{t=0}^{\infty} \beta^t \left(\ln[I_t - R_t] - A_t^* r_t{}^{\gamma} \left(\frac{I_{t-1}}{POP} \right)^{\mu} \xi_t \right)$$

where I_t is the number of people infected, R_t is the number of people who have recovered from Covid, r_t is the cost of the speed of infection, POP is population and ξ_t is a variable reflecting the varying infection-countering behaviour found in different sections of the population — which we will model shortly. The second term in the utility function is the cost to the virus both of a higher infection rate and of a rising lagged population share of infected people; both of these require the virus to work harder in terms of finding more cases to infect. $\left(\frac{I_{t-1}}{POP}\right)^{\mu}$ reflects special measures of protection taken by the government (lockdown etc.), as well as resistance rising with the expanding population share of infected people. t is days. $A_t^* = AV_t^{\chi}$ replaces A in our previous model, since now the vaccinated share of the population affects the effort required to infect the population.

The virus maximises this utility subject to the infection state model above, viz $I_t = r_t I_{t-1}$, or in logs, $\ln I_t = \ln r_t + \ln S_{t-1}$. The number of days over which the epidemic lasts is infinite because its progress is asymptotic, never reaching full infection of the population. As noted above, the virus needs to survive and so there must be infected people carrying the live virus for ever.

The first order conditions are simply found by creating the Lagrangean, while substituting the model of Recovered into the utility function and noting that $\ln(I_t - R_t) = \frac{1}{1-\rho} \ln I_t - \frac{\rho}{1-\rho}[\ln \rho + \ln I_{t-d}] = \frac{1}{1-\rho} \ln I_t -$

$\frac{\rho}{1-\rho}[\ln\rho + \ln I_{t-d}]$; the virus maximises, with respect to R_t and I_t the Lagrangean

$$L = \sum_{t=0}^{\infty} E_0 \left\{ \beta^t \left[\frac{1}{1-\rho} \ln I_t - \frac{\rho}{1-\rho}[\ln\rho + \ln I_{t-d}] - A_t^*[r_t]^\gamma \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu \right] + \lambda_t [\ln I_t - \ln r_t - \ln I_{t-1}] \right\}$$

to yield:

$$0 = \frac{dL}{dr_t} = -\beta^t A_t^* \gamma {r_t}^{\gamma-1} \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu - \lambda_t r_t^{-1}$$

whence:

$$-\beta^t A_t^* \gamma {r_t}^\gamma \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu = \lambda_t$$

Secondly, the first order condition w.r.t I_t yields:

$$0 = \frac{dL}{dI_t} = \beta^t \frac{1}{1-\rho} I_t^{-1} - \beta^{t+d} \frac{\rho}{1-\rho} I_t^{-1} - \beta^{t+1} A_{t+1}^* [r_{t+1}]^\gamma \xi_{t+1} \mu \left(\frac{I_t}{POP} \right)^\mu I_t^{-1} + (\lambda_t - \lambda_{t+1}) I_t^{-1}$$

and so:

$$\begin{aligned} 0 &= \frac{dL}{dI_t} = \beta^t \frac{1}{1-\rho} - \beta^{t+d} \frac{\rho}{1-\rho} - \beta^{t+1} [r_{t+1}]^\gamma \xi_{t+1} \mu \left(\frac{I_t}{POP} \right)^\mu A_{t+1}^* + (\lambda_t - \lambda_{t+1}) \\ &= \beta^t \frac{1}{1-\rho} - \beta^{t+d} \frac{\rho}{1-\rho} + \beta \frac{\mu}{\gamma} \lambda_{t+1} + (\lambda_t - \lambda_{t+1}) \end{aligned}$$

It follows that:

$$\left(1 - \left(1 - \beta \frac{\mu}{\gamma} B^{-1} \right) \right) \lambda_t = - \left[\beta^t \frac{1}{1-\rho} - \beta^{t+d} \frac{\rho}{1-\rho} \right]$$

where B^{-1} is the forward expectations operator leading the variable and keeping the expectations date given.

Hence

$$\lambda_t = - \left[\beta^t \frac{1}{1-\rho} - \beta^{t+d} \frac{\rho}{1-\rho} \right] / \left(1 - \left(1 - \beta \frac{\mu}{\gamma} \right) \right)$$

Now note that

$$\lambda_t = -\beta^t \gamma {r_t}^\gamma \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu A_t^*$$

then we have:

$$0 = -\beta^t \gamma {r_t}^\gamma \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu A_t^* = - \left[\beta^t \frac{1}{1-\rho} - \beta^{t+d} \frac{\rho}{1-\rho} \right] / \left(1 - \left(1 - \beta \frac{\mu}{\gamma} \right) \right)$$

or

$$\gamma {r_t}^\gamma \xi_t \left(\frac{I_{t-1}}{POP} \right)^\mu A_t^* = \frac{1}{(1-\rho)\left[1 - \left(1 - \beta \frac{\mu}{\gamma} \right) \right]} [1 - \rho\beta^d]$$

Finally in logs we obtain:

$$\ln r_t = \frac{1}{\gamma} \left\{ \ln \left[\frac{[1-\beta^d\rho]}{(1-\rho)\left(1 - \beta[1 - \frac{\mu}{\gamma}] \right)} \right] - \ln\gamma - \ln\xi_t - \mu \ln \left(\frac{I_{t-1}}{POP} \right) - \ln A_t^* \right\} \tag{1}$$

and so using $\ln I_t = \ln r_t + \ln I_{t-1}$

$$\ln I_t = \frac{1}{\gamma}\left\{\ln\left[\frac{[1-\beta^d\rho]}{(1-\rho)\left(1-\beta[1-\frac{\mu}{\gamma}]\right)}\right] - \ln\gamma - \ln\xi_t + \mu\ln POP - \ln A_t^*\right\} + \left(1-\frac{\mu}{\gamma}\right)\ln I_{t-1} \quad (2)$$

$$D_t = \delta I_{t-d}; R_t = \rho I_{t-d} \quad (3)$$

$$C_t = \Psi I_t \quad (4)$$

The model tells us that the daily infection rate responds inversely to the current self-isolation efforts of the population, ξ_t, and the existing (lagged) share of infected population, offsetting these in order to keep the costs of infection smooth over time, while still ensuring that the population gets steadily infected, ensuring new infections indefinitely.

We now insert household behaviour into the model. We will assume that household utility is reduced by infection but also by the personal inconvenience of avoiding infection by self-isolation activity, ξ_t. As this increases, the personal costs of not participating socially and economically rise directly with the extent of isolation, and rise indirectly the more uninfected people there are, as this lowers the personal risk of infection from participating, which raises the net costs of self-isolating (the economic costs net of the gain in lower infection risk). There is also a preference error, ϵ_t:

So

$$U_H = \sum_{t=0}^{\infty}\beta^t\left(-\ln I_t - \left[\xi_t\left(\frac{POP}{I_t}\right)^{\phi}\epsilon_t\right]\right)$$

Households maximise this utility with respect to ξ_t subject to the virus' behaviour set out above. Hence its Lagrangean is

$$L_{H0} = \sum_{t=0}^{\infty} E_0\left(\begin{array}{c}\beta^t\left(-\ln I_t - [\xi_t(\frac{POP}{I_t})^{\phi}\epsilon_t]\right)\\ -\nu_t[\ln I_t - \frac{1}{\gamma}\left\{\ln\left[\frac{[1-\beta^d\rho]}{(1-\rho)\left(1-\beta[1-\frac{\mu}{\gamma}]\right)}\right] - \ln\gamma - \ln\xi_t + \mu\ln POP - \ln A_t^*\right\} - \left(1-\frac{\mu}{\gamma}\right)\ln I_{t-1}]\end{array}\right)$$

Going through analogous Lagrangean steps to find the first order conditions yields from $0 = \frac{dL}{d\xi_t}$:

$$\nu_t = -\beta^t\gamma\xi_t^{\phi}\left(\frac{POP}{I_t}\right)\epsilon_t$$

and from

$$\begin{aligned}
0 &= \frac{dL}{dI_t} = -\beta^t I_t^{-1} + \beta^t\phi\xi_t^{\phi}\left(\frac{POP}{I_t}\right)\epsilon_t I_t^{-1} - \nu_t I_t^{-1} + \left[1-\frac{\mu}{\gamma}\right]\nu_{t+1}I_t^{-1}\\
&= -\beta^t + \beta^t\phi\xi_t^{\phi}\left(\frac{POP}{I_t}\right)\epsilon_t - \nu_t + \left[1-\frac{\mu}{\gamma}\right]\nu_{t+1}\\
&= -\beta^t - (\phi/\gamma)\nu_t - \nu_t + \left[1-\frac{\mu}{\gamma}\right]\nu_{t+1}\\
&= -\beta^t + \left[1+(\phi/\gamma) - \left[1-\frac{\mu}{\gamma}\right]B^{-1}\right](-\nu_t)\\
&= -\beta^t + \left[1+\phi/\gamma - \beta\left[1-\frac{\mu}{\gamma}\right]B^{-1}\right]\left\{\beta^t\gamma\xi_t\left(\frac{POP}{I_t}\right)^{\phi}\epsilon_t\right\}\\
&= -1 + \left[1+\phi/\gamma - \beta\left[1-\frac{\mu}{\gamma}\right]B^{-1}\right]\left\{\gamma\xi_t\left(\frac{POP}{I_t}\right)^{\phi}\epsilon_t\right\}
\end{aligned}$$

so that: $\gamma\xi_t(\frac{POP}{I_t})^{\phi}\epsilon_t = 1/\left\{1+\phi/\gamma+\beta[1-\frac{\mu}{\gamma}]\right\}$

$$\ln \xi_t = -\ln\left\{1+\phi/\gamma+\beta\left[1-\frac{\mu}{\gamma}\right]\right\} - \ln\gamma - \ln\epsilon_t - \phi\ln POP + \phi\ln I_t$$

When this is substituted into the infections equation we obtain:

$$\begin{aligned}\ln I_t &= \frac{1}{\gamma+\phi}\left\{\ln\left[\frac{[1-\beta^d\rho]}{(1-\rho)\left(1-\beta[1-\frac{\mu}{\gamma}]\right)}\right] + \ln\left\{1+\phi/\gamma+\beta\left[1-\frac{\mu}{\gamma}\right]\right\} + (\mu+\phi)\ln POP - \ln A - \chi\ln V_t + \ln\epsilon_t\right\} \\ &\quad + \left(\frac{\gamma-\mu}{\gamma+\phi}\right)\ln I_{t-1}\end{aligned}$$

This is the same equation as in our earlier paper with the exception that it now includes the effect of vaccination.

2 Results

The model is estimated and tested using the method of Indirect Inference (see Le et al. 2016 for more details). This is a simulation based method similar to simulated method of moments. To test the model an auxiliary model is used as a descriptor of the data and we then measure how close the parameters of the auxiliary model using the actual data are to the average of the parameters estimated from the simulated model. If the actual data coefficients are within the 95% bounds of the distribution of coefficients from the simulated data then the model is not rejected, and therefore the model is a good descriptor of the data. When estimated the structural model parameters the parameters are varied until a set is found with the highest p-value. This set of parameters would be the closest to the data. In this paper we use a logistic function as the auxiliary model, which takes the form:

$$f(x,a,b,c) = \frac{c}{1+e^{-(x-b)/a}}$$

where x is time, and the three parameters are:

a is the infection speed

b is the day when the maximum number of new infections occurred

c is the total number of recorded infected people at the end of the infection

These are the three parameters we try to match in the Indirect Inference procedure.

Table 1 shows the estimated structural parameters for the three Waves under consideration, whilst Table 2 shows the logistic function parameters for the actual data along side the mean and confidence bounds from the simulations.

	Wave 1	Wave 2	Waves 3-4
γ	74.8847	68.9229	85.5612
μ	3.3705	3.5940	5.3381
α	−9.3384	−5.1107	−8.4966
ϕ	0.1457	0.3192	0.9270
μ/ϕ	23.1	11.3	5.8
χ	NA	0.1947	0.7610
$(\mu+\phi)/(\gamma+\phi)$	0.047	0.056	0.071
Wald	0.2972	8.9507	10.6825
P-value	0.9639	0.0827	0.0574

Table 1: Structural Model Parameter Estimates for the Three Waves

Wave 1	Actual	Lower 2.5%	Upper 2.5%	Mean	IN/OUT
a	8.8239	0.2840	33.4295	9.8216	IN
b	65.2579	41.3138	135.0159	73.5042	IN
c	4464493.4789	2164690.1735	16852446.2495	6608217.7604	IN
Wave 2					
a	31.1942	3.0204	51.5670	13.6528	IN
b	172.3836	39.1620	148.4174	70.8923	OUT
c	18625762.1456	7610184.5821	20037886.5767	16071419.0855	IN
Waves 3–4					
a	34.5802	2.1590	38.8036	8.3632	IN
b	141.2116	29.2303	104.9454	48.7938	OUT
c	19821214.5578	4626993.9865	20072732.2153	14350683.6932	IN

Table 2: Auxiliary Model Parameter Bounds for the Waves

2.1 Comments on UK results:

We find that Waves 1 and 2 have virtually the same parameters. In both the lockdown factor, μ, is a large multiple of the personal response factor, ϕ, reflecting large lockdown interventions in both. With the third-fourth wave the estimates change sharply, reflecting the vaccine roll-out. γ, the degree of immunity rises sharply; μ also rises as resistance to the virus increases with higher penetration, due to so many being vaccinated. Also ϕ rises with people responding much more to increased penetration, being made more confident by the vaccine. The vaccine penetration itself has of course a direct effect on the virus' progress, absent in previous waves.

The three Waves differ in the overall numbers infected as shown in Table 3:

	Actual c	Model Mean c	Model Steady-state	Data
Wave 1	4.5	6.6	4.7	4.7
Wave 2	18.6	16.1	18.2	18.2
Wave 3	19.8	14.4	17.3	17.3

Table 3: Table of Results for Total Infected (millions)

We can see here that the logistic c value is reasonably matched by the model mean, while the steady state of the model is constrained in estimation (via the constant) to equal the data total. It is striking how many fewer were infected in the first wave than in the second two, where about four and a half times as many were infected in total.

Deaths however were about equal in total in the first two waves: this underlines how high the initial death rate was and how much it fell in the second wave (by a factor of 4). The death rate fell steadily across all waves, falling to a far lower rate from the third wave with vaccination as can be seen in Figure 1.

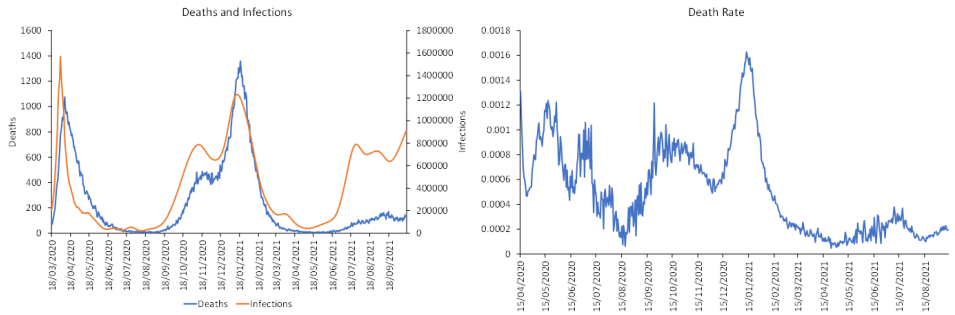

Figure 1: Deaths and Infections

2.2 Analysing trends in the hospitalisation and death rates of the UK:

Figure 2 shows the evolution of two ratios: deaths to hospitalisations and hospitalisations to infections, where hospitalisations are measured by those in hospital. The final plot in Figure 2 shows the number of people estimated to have Covid. In all ratios we can see that even though the estimated number infected is increasing as we progress through the waves, the ratios are decreasing.

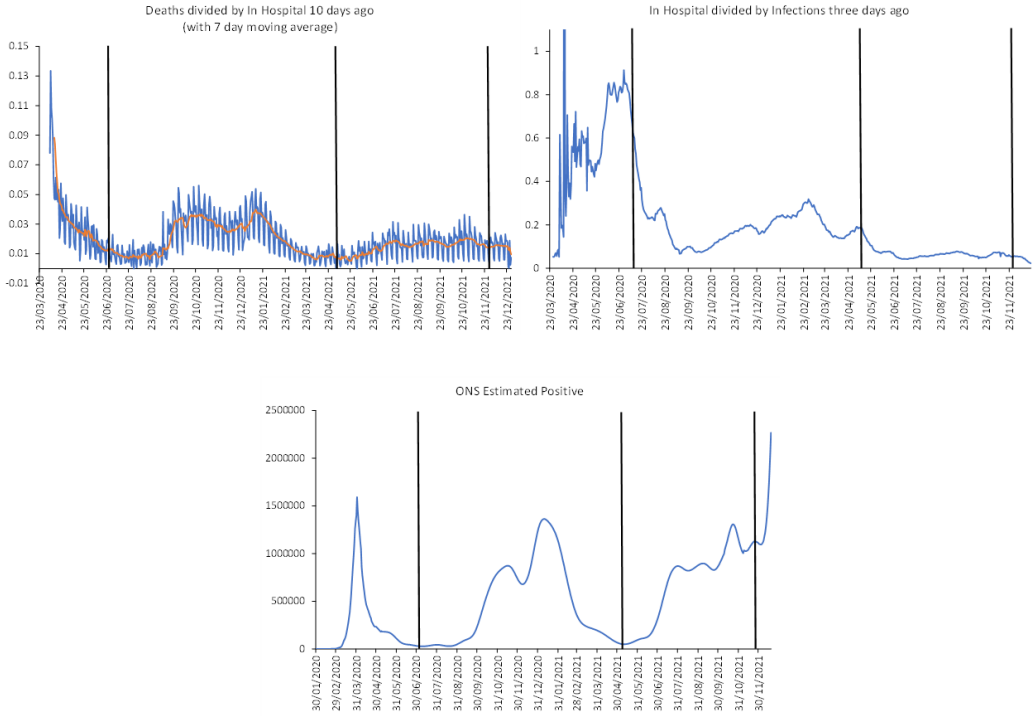

Figure 2: Data on Infections, Hospitalisation and Deaths over the 4 Waves

The key question as we move forward into Wave 4 is how the hospitalisation and death rates will evolve. If these are disengaged from the infection rate, then it becomes possible to continue 'living with Covid'. If however they remain high enough to precipitate excessive numbers of hospitalisations and deaths, then further lockdown interventions will be forced back into the agenda.

We examine this by regressing hospitalisation/cases on the double-vaccination rate and the cumulated number infected as percent of the population ($PCINF$, to proxy rising immunity) and the percentage of the population fully vaccinated ($VACC$, a weighted average of the percentage of the population who have had either 2 or 3 vaccinations). We then do the same for deaths/lagged hospitalisations; here $PCINF$ will also pick up the effect of the better treatments that have emerged with the experience of infection. For the Wave 4 numbers we may also find an effect of the rising booster rate.

To discover what might be driving the trends in the virus' behaviour we took the detailed UK data on estimated cases, hospitalisations and deaths and regressed the hospitalisation ratio to lagged infections and the deaths ratio to lagged hospitalisations to determine the roles of vaccines and immunity on the evolving figures. We would expect that vaccines would have a steady effect but that immunity would have an increasing effect as the virus aged.

Figure 3 shows the percentage of the population that have Covid and vaccinated.

We found, in Table 4, that there are cointegrating relationships to both the hospitalisation (i.e. those in hospital) ratio to infections and the death ratio to hospitalisations from the vaccination rate and the overall past total infection rate, proxying the resulting immunity. We find also a clear error-correcting equation relating the change in these series to the current shocks to vaccination (negative) to current infections (positive) and the lagged deviation from trend (negative); all of these effects are significant. However, these

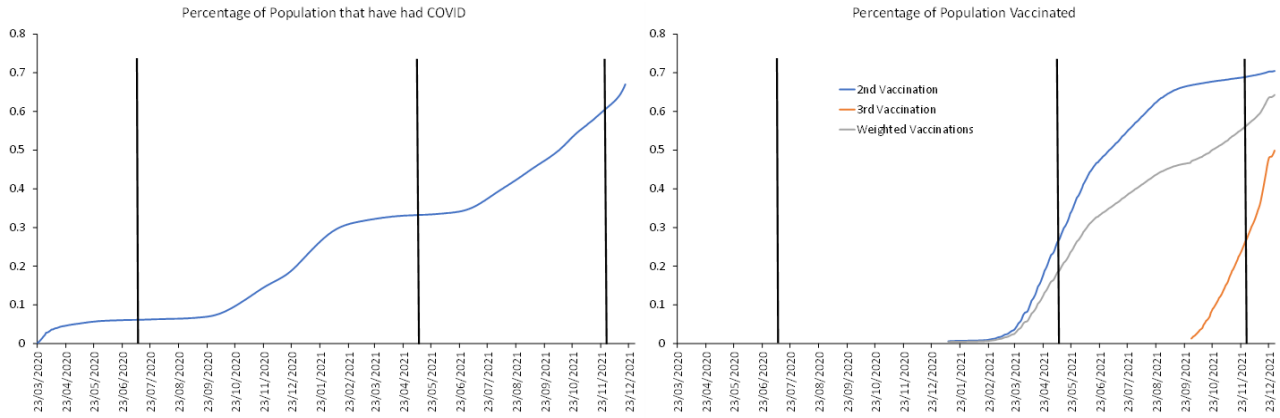

Figure 3: Vaccinations and Infections

regressions suggest that vaccinations, when the various vaccine elements are weighted together to create a meaningful vaccine variable, were less important than immunity (proxied by the cumulative total/population of those infected, $PCINF$) in reducing the trends in hospital/cases and in deaths/those in hospital. The $VACC$ weighted variable is insignificant in both cointegrating regressions while $PCINF$ is significant and rightly signed in both. This trend effect in $PCINF$ is picking up the early tendency of hospitalisation and deaths to fall well before vaccination started. Nevertheless, both variables have significant short run impacts as well. So both play an important role.

	IHI3	DIH10
Long Run Relationship		
Constant	0.387331^{***}	0.025383^{***}
$VACC$	-0.048444	0.002388
$PCINF$	-0.639231^{***}	-0.023662^{***}
ECM Regression		
Constant	0.017141	-0.002265^{***}
$\Delta(VACC)$	-3.593752	-0.717900^{**}
$\Delta(PCINF)$	-13.17548	2.803055^{***}
Long Run Residual(-1)	-0.482439^{***}	-0.393575^{***}
Cointegrating ADF p-value	0.003666	0.023863

IHI3=(In Hospital)/Infections(-3), DIH10=Deaths/(In Hospital(-10))
***p<0.01, **p<0.05, *p<0.10

Table 4: Trends in the Virus Behaviour

These regressions suggest that vaccinations were less important than immunity in reducing the trends in hospital totals and deaths. The $VACC$ weighted variable is insignificant in both cointegrating regressions while $PCINF$ is significant and rightly signed in both.

3 Data and results for Sweden:

3.1 Data

The number of reported cases from the Swedish Health Agency and deaths reported via Johns Hopkins are shown in Figure 4.

It would seem that, given the likely death rate and the deaths data, the Swedish cases are a considerable

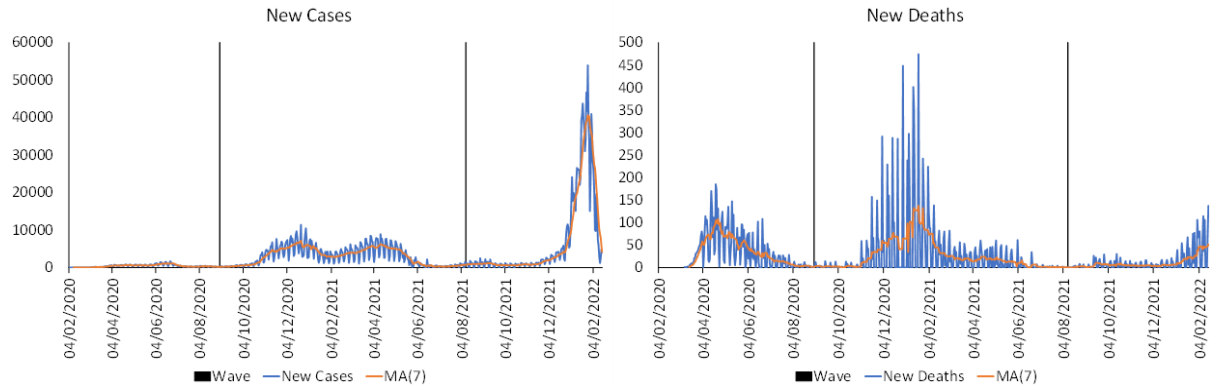

Figure 4: Swedish COVID cases and deaths

underestimate of infections. We therefore use the death rates for the UK to estimate Swedish infections. Total deaths/total infections for each UK wave are in Table 5.

	Start Date	End Date	Death Rate
Wave 1	30/01/2020	05/07/2020	0.008542
Wave 2	06/07/2020	08/05/2021	0.004769
Wave 3	09/05/2021		0.000863

Table 5: Death Rate for UK Waves

If we apply these to Swedish deaths from Covid we obtain Figure 5:

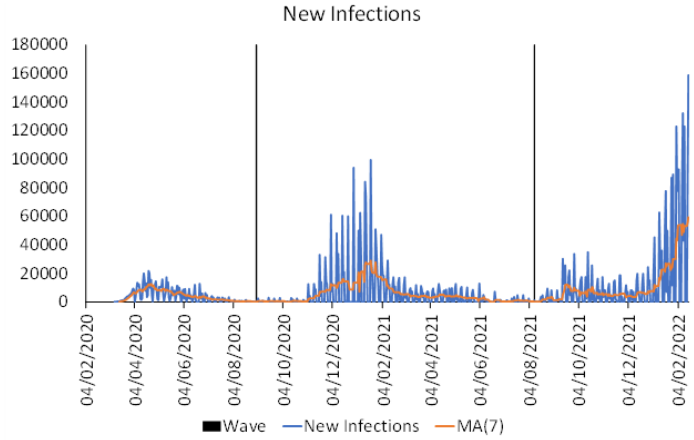

Figure 5: Estimated Swedish COVID infections

This gives a total infected over all waves of 5,126,659, approximately half the population; this compares with around 65% for the UK and is therefore of a plausible order of magnitude. It is this infections data that we use for our Swedish estimates.

Finally, for vaccinations we use data from the Our World in Data database (Mathieu et al., 2021). Weekly data is available from 03/01/2021–06/02/2022, and the data available at irregular frequency after this date, therefore we have interpolated the data to a daily frequency as shown in Figure 6.

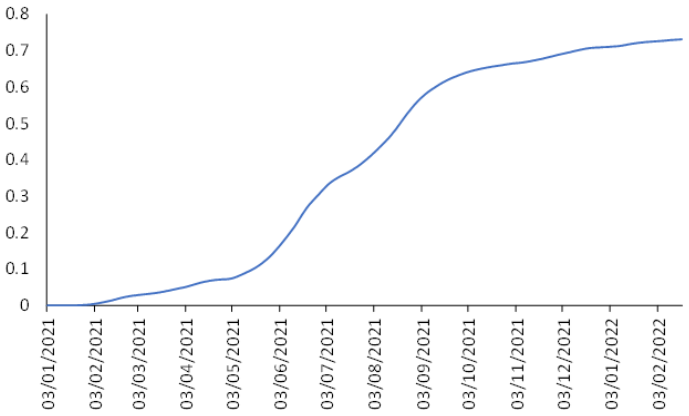

Figure 6: Fraction of Swedish population fully vaccinated

3.2 Estimates of Swedish model

After estimating the model for the four waves we found that the model could not fit for Waves 3 and 4. A possible explanation for this is that the data for these waves do not look S-shaped as in a logistic function; in effect these waves look most like the start of on uncompleted wave and hence do not fit our auxiliary model which applies to a fully completed wave. The structural parameters are shown in Table 6, and the auxiliary model parameters in Table 7. For Wave 1 we found that the model fits very well with a P-value of 0.458, and all of the auxiliary model parameters are within the 95% bounds from the simulations and very close to the mean. For Wave 2 the models also fits well with a P-value of 0.1554, though it slightly over predicts the c coefficient of the auxiliary model.

	Wave 1	Wave 2
γ	149.0657	138.8889
μ	6.3528	1.4370
α	−19.1283	−2.2917
ϕ	0.7525	0.2541
μ/ϕ	8.44	5.65
χ	NA	0.1445
$(\mu+\phi)/(\gamma+\phi)$	0.047	0.011
Wald	2.3474	5.2320
P-value	0.4580	0.1554

Table 6: Structural Model Parameter Estimates for the first Two Waves for Sweden

Wave 1	Actual	Lower 2.5%	Upper 2.5%	Mean	IN/OUT
a	17.7081	8.8439	21.6103	13.9386	IN
b	57.7389	47.4391	66.7075	54.9981	IN
c	685587.4326	603729.3359	825439.7868	697683.8315	IN
Wave 2					
a	39.5683	33.7709	56.8983	44.3651	IN
b	53.3256	48.2126	78.9809	61.9125	IN
c	2669698.6920	2742545.9734	3451924.9276	3104045.5320	OUT

Table 7: Auxiliary Model Parameter Bounds for the Waves, Sweden

In our previous estimates (Meenagh and Minford, 2021) for Sweden (replicated in Table 8), which were based on deaths and were directly comparable with our UK estimates, also based on deaths, we found that

the lockdown parameter for the UK was much larger than Sweden's while the personal response parameter, ϕ, was much smaller; the ratio of the lockdown to the personal response parameter, μ/ϕ, was therefore much lower in Sweden than in the UK. In our latest estimates for Sweden the Swedish ratio of lockdown to personal response is also lower than the UK's in both waves, averaging about 7 compared with 17, which tallies with our earlier result; it is also similar to the UK's wave 3/4 (at 5.6) which tallies with the UK's policy shift away from lockdown to vaccination. So on the central policy issue of the use of mandatory relative to advisory intervention the latest results do cohere with our earlier ones and with what we know about the two countries' policies.

	UK	Sweden	Global
δ	0.0084	0.0052	0.0015
μ	4.11	0.151	2.55
γ	59.53	40.59	79.02
ϕ	0.17	2.95	0.62
μ/ϕ	24.2	0.05	4.11
$(\mu+\phi)/(\gamma+\phi)$	0.07	0.07	0.04
Constant	−11.94	−8.605	−10.36
% Population Infected to Date	7	7	7
% Population Infected Long Term	7	7	7
Reported/Actual Infections (inverse)	0.0499(20)	0.0442(23)	0.0337(30)
P-value	0.93	0.82	0.70

Table 8: Structural Model Parameter Estimates

4 Economic costs of Covid policies

We noted in our previous paper that Sweden's Covid policies based on less direct intervention and more advice for personal behaviour caused much less economic cost in the first wave while leaving the course of infection virtually the same. We find the same here with our updated estimates. Sweden's less interventionist approach in both Wave 1 and Wave 2 resulted in a much smaller loss of GDP relative to its pre-Covid level, as shown in Figure 7. The UK's approach abandoned lockdown in favour of vaccination and advice in waves 3/4, to conform to Sweden's.

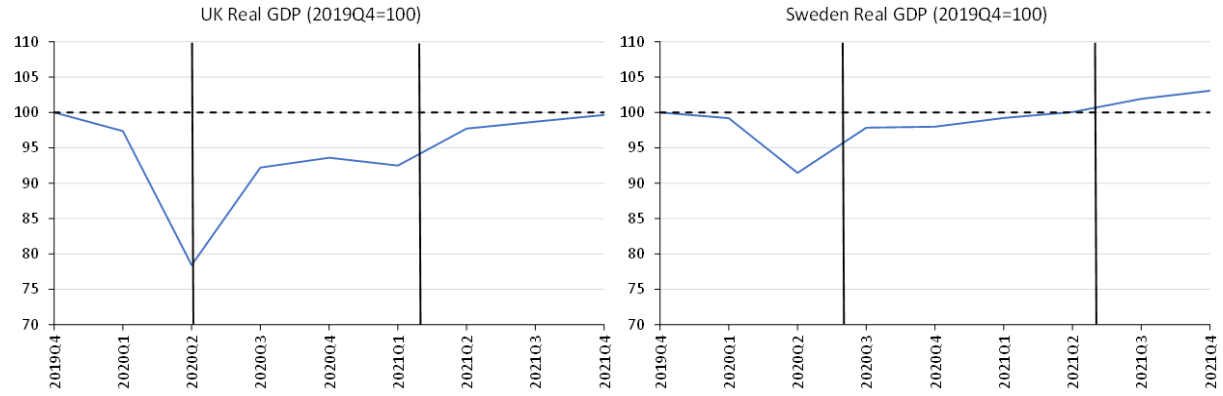

Figure 7: Real GDP during the COVID waves

Again, the updated pattern of the two countries' cumulative infections is hard to distinguish, as Figure 8.

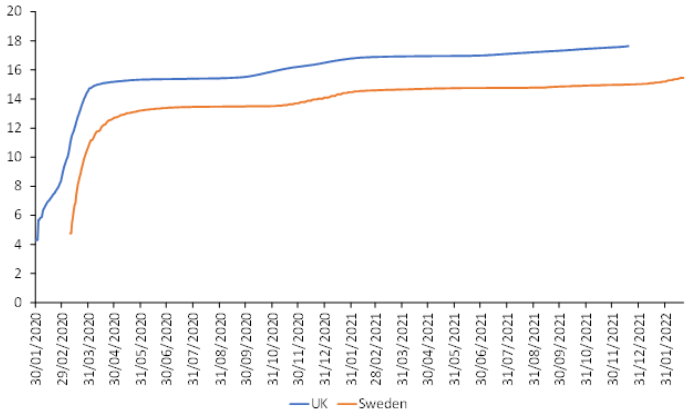

Figure 8: Cumulative Infections (in logs) for UK and Sweden

What we see is that on average in Wave 1 UK GDP was down 8.5% on pre-Covid (2019 Q4) versus 3.8% for Swedish GDP; and in Wave 2 UK GDP is down 8.5% vs Sweden being down 1.1%; in Wave3 UK GDP was 1.7% below the pre-Covid level, whereas Sweden recovered to 2.2% above the pre-Covid level. Thus, weighting each wave equally, the average loss from UK policy vs Sweden's was 5.3% over the pandemic as a whole.

5 Conclusions

In this paper we have used the model of Covid transmission set out in Meenagh and Minford (2021) to extend our empirical estimates to the fuller data on the pandemic up to the end of 2021 by which time there had been four waves of infection in the UK. We used the newly available sample-based estimates of infections created by the ONS in place of infections estimated from deaths that we used before. These new and reliable estimates allowed us also to relate the progress of the disease, including its hospitalisation and fatality rate, to vaccination and population immunity due to prior infection. Finally, we updated our comparison with Sweden for the most recent data. Unfortunately, there is no equivalent sample-based data for infections there — as the ONS sample survey approach has not been carried out anywhere else in the world to our knowledge.

Our updated results are much in line with our earlier ones: the Swedish lockdown intensity relative to personal response was well below the UK's in the first two waves and resulted in much lower economic costs while leaving the course of the disease hardly altered. By wave 3/4 the UK had moved away from lockdown and its estimated relative intensity fell with it, accompanied by lower economic costs.

Our unique UK sample-based data led us to find that the most significant factor driving the trends in hospitalisation and deaths was the population's accumulated infection rate, a measure of its herd immunity. The vaccination rate also had a significant short run effect on the fatality rate.

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